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AND THE YEARS AHEAD

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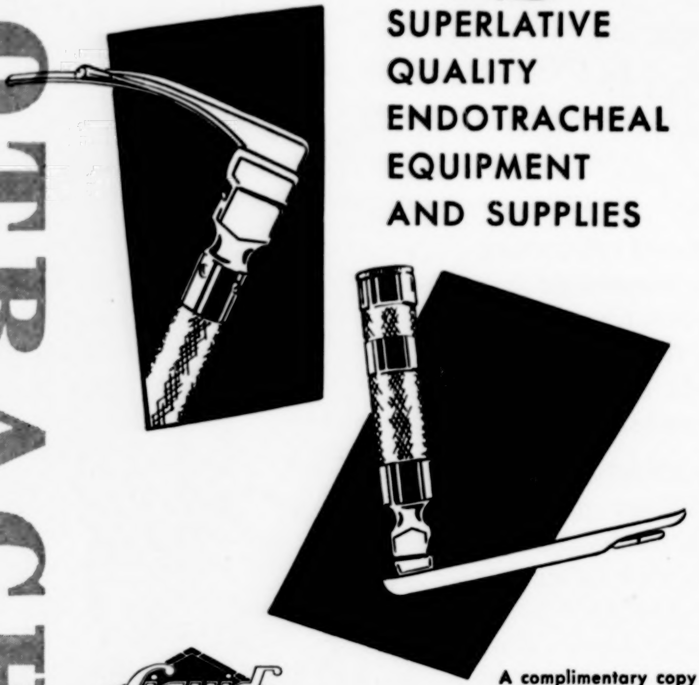

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REFERENCES:
1. Report of the Council on Pharmacy and Chemistry of the A.M.A.: In press. **2.** Hudon, F., and Jacques, A.: Proc. Canad. Anesthetists Soc. (1952) p. 34. **3.** Orkin, L. R., and Rovenstine, E. A.: Anesthesiology 13:465 (Sept.) 1952. **4.** Crawford, O. B.: Anesthesiology 14:278 (May) 1953. **5.** Lee, L. W., Davis, E., Jr., and Barmore, J.: To be published. **6.** Arcuri, R. A., Newman, W., and Burstein, C. L.: Anesthesiology 14:46 (Jan.) 1953. **7.** Hudon, F., and Gendron, P.: Laval med. 18:145 (Feb.) 1953. **8.** Ray, E. S., and Vinson, P. P.: Anesthesiology 14:315 (May) 1953. **9.** Clark, R. E., Orkin, L. R., and Rovenstine, E. A.: To be published. **10.** Stark, E. J.: To be published. **11.** Proctor, A. M., et al.: Paper presented at 11th Conference on Chemotherapy of Tuberculosis, V. A., St. Louis, 1952.

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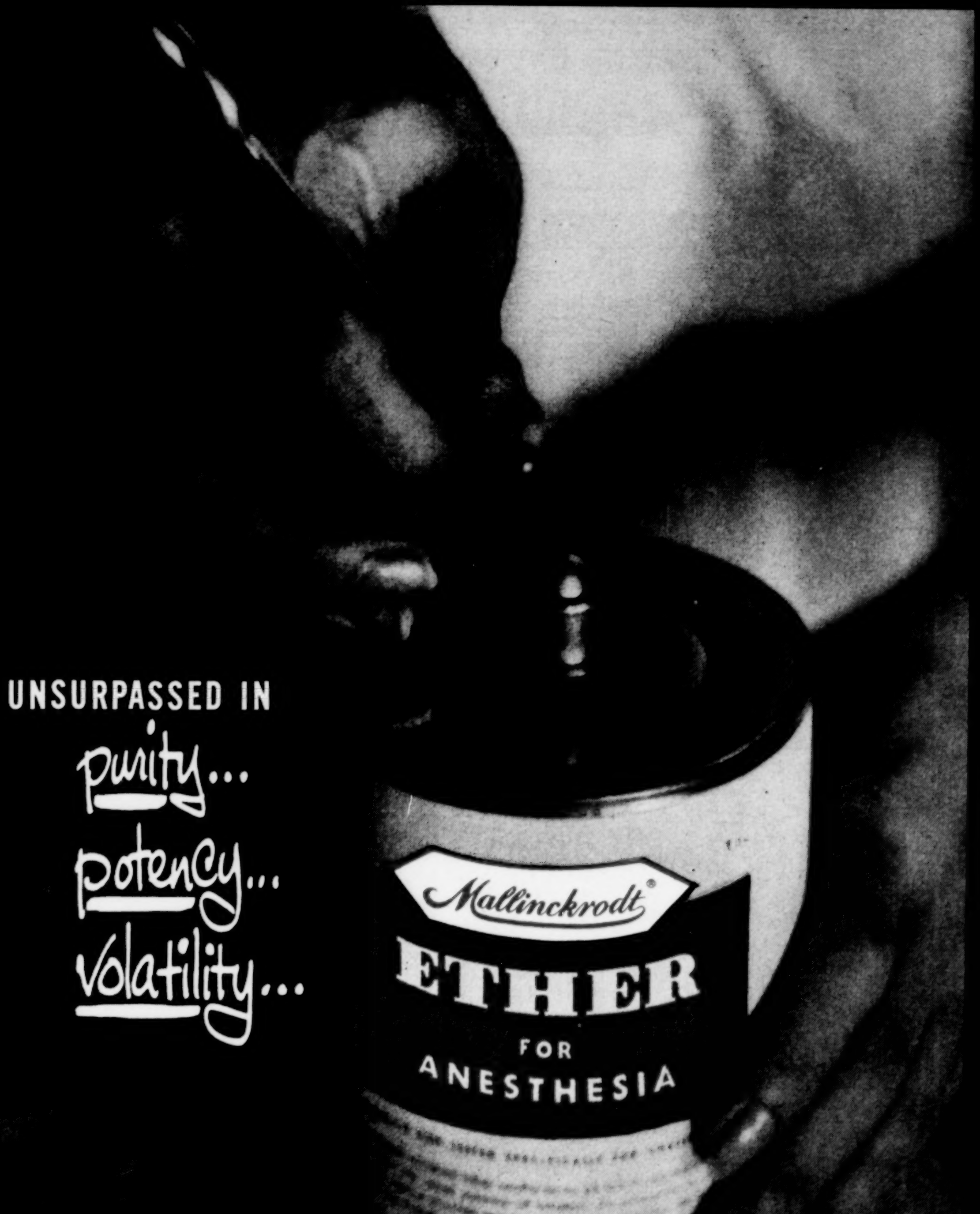
The Weakest Link

"A.A.N.A. is only as strong as its weakest link" is an adaptation of an old copy book maxim that merits serious thought by each of us. Our organization is made up of approximately seven thousand links (members). Most are strong, in that they are of a high caliber of professional integrity. Some are weak, because they maintain a narrow, "personal gain," attitude. The strong links are building for the profession of Nurse Anesthetists a place that is secure and enviable. The weak links are taking advantage of all that has been established and, at the same time, undermining and deterring the building.

Who are these weak links? They are those among us who, apparently, are working for personal advantage only, making unreasonable demands on hospital administrators and patients. Exorbitant salaries and short hours are paid for by the patient, often to the embarrassment of the administrator, who must raise rates in order to meet the demands of certain anesthetists. Some weak links are arrogant and dictatorial with the surgeons, ignoring the fact that, if understanding and compromise cannot be reached amicably between the surgeon and the nurse, the opinion of the surgeon must prevail, since his is the ultimate responsibility. The weak links are those of us who seem to forget that, although we have had a year or more of special training and have been entrusted with work that entails great responsibility, we are still nurses and should not isolate ourselves from other nurses, nor express an attitude of "better than thou" toward them. Other weak links stay on the sidelines to criticize and question all that is done by those who, in addition to having the same busy daily schedule, give freely of time, thought and effort to build and maintain for us all a place that is strong and secure.

A weak link here and there along the chain can jeopardize the entire work of the predominantly strong links. Let us look to our professional ethics, move out of the weak link class, and at least support the work of the strong links, if we do not wish to contribute to it.—

Minnie V. Haas, R.N.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1. Brown, S., and Fehlman, B. F.: Missouri Med. *50*:609 (Aug.) 1953.
2. Stephen, C. R., Nowill, W. K., and Martin, R.: Anesthesiology *13*:646 (Nov.) 1952.
3. Flowers, C. E., Jr.: Am. J. Obst. & Gynec. *65*:1027 (May) 1953.

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Hope for an Age of Analgesia

John S. Lundy, M.D.*
Rochester, Minnesota

On the morning of July 30, 1953, I had the opportunity to administer dolitrone (previously MRD-125)[1;2] intravenously to a volunteer male subject. Of course, I gave it very slowly to begin with and often I asked the subject how he felt. He replied that he had a feeling of inebriation. When the dose still was relatively small, I noticed that pricking the skin of his forearm with pins did not cause him to move his arm. This led me to hope that a small dose of this drug would produce cutaneous analgesia. Finally the dose became sufficiently large to produce anesthesia.

That afternoon I used the drug in 2 cases of dental extraction. During the operations these patients would open their eyes or would swallow on command, yet afterward they had no memory of what had been done. Very shortly after administration of the drug had been discontinued, each of them was fully awake and could put his finger on his nose, or nearly on his nose. From then on, when I could obtain the drug, I continued to use it for extraction of teeth and for other operations. I now have used it in 130 cases.

The state just described was not new in my experience. Occasionally in the previous 30 years I had produced it by administering, in the same case, reduced quantities of several drugs. Always I had sought the state, primarily because small doses meant safety for the patient. With the use of dolitrone, I recognized a quality of this much-sought state that was superior to what I had experienced and I tried to think of a name for it. The best I could do was to call it by a name which means various things to various people. I called it "analgesia" or "general analgesia[3]." If anyone can come up

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[1] Anesthetics, Local and General. Chemical & Engineering News. 32:1323 (Apr. 5) 1954.

[2] Lundy, J. S.: 110 Years of Anesthesia. J. M. A. Georgia. 43:195-200 (Mar.) 1954.

[3] Lundy, J. S.: Will There Be an Era of Analgesia? (Editorial.) Journal-Lancet. 74:155 (Apr.) 1954.

Read at the meeting of the American Association of Nurse Anesthetists, Chicago, Illinois, September 16, 1954.

*Section of Anesthesiology and Intravenous Therapy, Mayo Clinic and Mayo Foundation. The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

with a better term, all who are concerned with the subject will be grateful.

In the opening paragraph, it was said that when the dose of dolitrone was increased, anesthesia resulted. Thus, anesthesia can be produced with this drug alone but the dose can be greatly reduced if hyatrobal and methadone hydrochloride are given before operation, and if a mixture of 80 per cent nitrous oxide and 20 per cent oxygen is administered during the operation. Nitrous oxide is definitely analgesic and dolitrone in a small dose is analgesic. Any nausea that might follow use of dolitrone is forestalled by the hyatrobal, and the methadone hydrochloride improves the quality of the analgesia. Generally speaking, when this combination of drugs has been administered for dental extraction analgesia has been of approximately 1 minute's duration for each 1.5 cc. of a 2.5 per cent solution of dolitrone. The largest dose that I have used has been 1875 mg. in 2 hours. In this case an abdominal operation was performed. A feature I had not experienced before was that the normally tubular intestine seemed to be flat and peristalsis seemed to be absent. The bowel was packed off into the upper part of the abdomen and did not protrude into the lower part until the pack was removed. This was the more remarkable because the abdominal muscles were so poorly relaxed that a self-retaining retractor was used to separate them satisfactorily.

A dental surgeon told me that he believed there was less bleeding when dolitrone was used than when such agents as ether, cyclopropane, ethylene or pentothal sodium were employed. Seldon[4] obtained some evidence to support this by observing the capillaries in the rabbit's ear through a Clark window. He found that when dolitrone was used the capillaries were smaller than when other agents which cause slight capillary dilation had been used. Unless preliminary medication had been given, the skin temperature of the legs of patients who were undergoing dental extraction did not increase when dolitrone was used, as it would have done if the capillaries had dilated.

In a few cases I have used dolitrone in producing anesthesia for hemorrhoidectomy. Of the first 6 patients, each of 4 required 1 injection of morphine postoperatively and 2 required none. Five of these patients had been given hyatrobal and methadone hydrochloride as preliminary medication and 1 had received pentobarbital sodium (nembutal sodium) 3 grains (0.2 gm.) and morphine sulfate 1/6 grain (0.01 gm.). The dolitrone was given with the patient lying face down and with the hips elevated on a kidney rest. Because the anal sphincter did not relax, 2 cc. (6 gm.) of solution tubocurarine chloride was administered intravenously; relaxation was then satisfactory. Why these patients were so comfortable after operation is not clear and this effect must be investigated.

---

[4] Seldon, T. H.: The Effect of General Anesthetic Agents on Small Blood Vessels. Thesis, Graduate School, University of Minnesota, 1940.

Collateral effects of dolitrone are largely favorable. It has but slight effect on blood pressure or respiration. The electrocardiogram gives evidence of only very little change in cardiac activity. When a very small dose of the drug has produced analgesia, electro-encephalograms resemble somewhat those produced by pentothal; when a large dose has produced surgical anesthesia, the pattern resembles that produced by cyclopropane. If intubation is necessary in a case in which anesthesia has been brought about by administration of dolitrone, laryngospasm is not likely to occur when the epiglottis has been picked up by the laryngoscope.

At first, the weakly acidic, powdered drug was dissolved in 1:500 solution of sodium hydroxide to make a 2.5 per cent solution, the pH of which was 11.4. This is more alkaline than a 2.5 per cent solution of pentothal sodium, of which the pH is 10.4. The second shipment consisted of a 10 per cent suspension of the drug in polyethylene glycol. As compared with the powder alone, the suspension went into solution much more rapidly. This solution, moreover, when diluted until it is as weak as 0.25 per cent, will remain stable for 2 to 4 hours; thus it can be administered by intravenous drip.

It has been possible to inject the suspension directly into the blood stream, without putting it into solution first. Since this can be done use of an alkaline solvent may not be necessary. Moreover, a technic has been worked out for intramuscular injection of the suspension. This opens new possibilities for use of dolitrone. Experience with intravenous and intramuscular injection of the suspension is very limited. Enough is known of these procedures, however, to show that the effect of one is unlike the effect of the other and that the effect of either differs from that obtained when the drug is injected intravenously in solution.

My initial experience with the drug indicated that it might be an unusually efficient cutaneous general analgesic. I hoped, then, that it would be useful in relieving the pain of burned patients. Now Dr. Paul H. Lorhan, of the University of Kansas Medical Center, Kansas City, Kansas, has written to me about a girl 3 years of age, 25 per cent of whose body sustained third degree burns on March 29, 1954. For changing the dressings, cyclopropane was used once without employment of an intratracheal tube and once with use of such a tube. Trichlorethylene proved unsatisfactory as an analgesic. Then dolitrone was administered intravenously with entire satisfaction; pain was allayed and the child was able to straighten her leg on command.

Also, Dr. Lorhan and I used dolitrone in one case of fracture. A woman, 73 years of age, who had a fractured hip, also needed some teeth extracted while she was in hospital. While analgesia was being induced, the patient remarked that for the first time the pain in her hip was gone. She required much more of the drug for extraction of the teeth than for relief of the pain in her hip.

In this brief presentation, I have spoken most of a particular preparation, dolitrone. That is a trade name. There is no other name for the preparation as yet. I believe it is a good preparation. Nevertheless, and I spoke of this earlier, my mind has been less on a particular drug than on 30 years during which I have hoped to administer various therapeutic substances in such combinations that the dose of each could be decreased, the safety of the patient increased and his consciousness retained, at least in part. I have not been alone in this hope. A few persons as old as I, and many younger, have shared the hope. Some of them have been surgeons, some nurse-anesthetists, some pharmacologists, some pharmaceutical manufacturers, and some anesthesiologists. If, as seems possible, the introduction of dolitrone has introduced, also, the long-sought age of what, thus far, must be called "analgesia" or "general analgesia[5]," we all shall gain, but greatest gainer of us all will be, as he should be, the patient in our hands.

---

[5] Lundy, J. S.: After a Century of Anesthesia, Will Analgesia Take Over? Unpublished data.

Controlled Induced Hypotension

Donald E. Hale, M.D.[*]
Cleveland

Controlled induced hypotension is used for four purposes: (1) to reduce blood loss and conserve a patient's blood, (2) to provide a clear field unobstructed by blood, (3) to reduce brain edema, and (4) to reduce the tension in large vessels which are being operated upon.

The conservation of blood is desirable in any operation in which blood loss may be sufficient to demand replacement by transfusion. It was a problem even before the advent of elective surgery. The stanching of hemorrhage by various means was practiced by the ancients. The tourniquet, boiling oil, and finally the ligature represented advances in the control of hemorrhage.

The need for saving blood is indicated in the following table which gives the average blood loss in some of the standard operations as reported by several different authors.

Operation	Blood Loss (cc)
Craniotomy	803
Radical Mastectomy	690
Thoractomy	1558
Operation on bile ducts	460
Nephrectomy	484
Hysterectomy	358

The conservation of blood is especially important in extensive operations in which even the exercise of great care does not prevent the loss of considerable blood. Such an operation is the pelvic exenteration for malignancy of the uterus.

The clear field is especially desirable in intracranial procedures and in the fenestration operation Among the intracranial lesions, olfactory groove meningoma and intracranial aneurysm are conditions indicating controlled hypotension. The olfactory groove meningoma obtains its blood supply from below, a site inaccessible to the surgeon until the growth has been removed. The intracranial aneurysm may be ruptured suddenly during operation and flood the whole field with blood which completely obscures the bleeding point.

The reduction of brain edema is of considerable value whenever it is encountered during intracranial surgery.

The dissection about a patent ductus arteriosus is greatly facilitated by induced hypotension.

Hypotension is induced by one of three methods: arteriotomy, spinal anesthesia, or the use of ganglioplegic drugs.

Read before the Twenty-first Annual Meeting of the American Association of Nurse Anesthetists, Chicago, September 16, 1954.

[*] Department of Anesthesiology, The Cleveland Clinic Foundation, and the Frank E. Bunts Educational Institute, Cleveland, Ohio.

Blood pressure depends upon (1) cardiac output, (2) peripheral resistance, (3) blood volume, (4) blood viscosity, (5) vessel elasticity. The first three of these factors can be modified artificially in order to reduce blood pressure during surgery. Blood pressure is maintained under normal circumstances and during periods of stress chiefly by the cardiac and vascular reflex mechanism. Impulses from the carotid sinus and aortic arch, and also depressor fibers from the vagus nerve, send impulses to the cardioinhibitory center, the cardio-accelerator center, the vasodilator center, and the vasoconstrictor center, all of which are located in the medulla. These centers, in turn, send impulses to the heart and blood vessels. Those which go to the heart control the rate and force of the contractions, while those to the vessels control their caliber. Thus, these two inter-related mechanisms help to keep the cardiac output and the capacity of the vascular tree in such relationship that blood pressure is maintained in spite of loss of blood volume.

Blood pressure is reduced by deliberately causing a discrepancy between the blood volume and the capacity of the vascular tree. The vascular tree and the blood volume may be likened to a toy long-necked balloon filled with fluid which extends up into the neck. The level of the fluid in the neck of the balloon can be reduced either by thinning out the wall of the balloon, which will reduce its elasticity, or by withdrawing some of the fluid from the balloon. The former situation is analogous to hypotension by vasodilatation, and the latter to hypotension by arteriotomy.

There is a regular downward gradient of blood pressure as the blood passes from the arteries to the arterioles to the capillaries and to the veins. The greatest drop of blood pressure occurs as the blood passes through the arterioles (from about 100 to about 30 mm. of mercury). In the capillaries the pressure falls from 30 to 10, and in the veins it falls to zero or below. In arteriotomy hypotension, however, the blood pressure falls from the level in the large arteries to a very low value—perhaps 5 mm. of mercury in the arterioles, which greatly decreases tissue perfusion. On the other hand, in vasodilatation the mean pressure in the arteries falls to a low level, and this is maintained fairly well throughout the arterioles and capillaries.

Arteriotomy[1] is carried out by introducing a two-limbed cannula into the radial artery. One limb of this cannula is connected by a rubber tube to a bottle containing ACD solution in which blood is collected. Through the other limb the blood is reintroduced into the artery of the patient during surgery, if needed, or otherwise at the end of the procedure. The patient is thus given an arterial transfusion of his own warm, well-oxygenated blood whenever necessary to combat a dangerously low blood pressure. The effectiveness of arterial infusion can be demonstrated by the injection of a radiopaque material into the brachial artery of a dog in which the blood pressure has been reduced to 20 mm. of mercury by bleeding. After the injection of 10 cc. of this fluid an x-ray demonstrates complete filling of the coronary arteries, the carotid arteries and the vertebral arteries, thus showing that such an arterial infusion immediately restores circulation to the myocardium and to the brain.

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[1] Gardner, W. James and Hale, Donald E., Arterial Bloodletting During Operation As Aid In Hemostasis, The American Journal of Surgery, Vol. LXXIX, #5:635-644, May 1950.

Hypotension by vasodilatation may be accomplished by spinal anesthesia,[2] by ganglion block,[3] or by agents which act directly on blood vessel musculature. Spinal anesthesia causes preganglionic block by anesthetizing the spinal nerve filaments before they coalesce into the posterior spinal nerve root. Ganglionic block is carried out by use of hexamethonium. Arfonad,[4] which has some ganglion blocking effect also, is used to cause paralysis of the muscle in the blood vessel wall.

The dangers of hypotension are the result of the effect of low blood pressure upon different body systems. Cardiac arrest readily results if the myocardium is deprived of its oxygen supply for short periods of time. Decortication of the cerebrum follows brain anoxia of three to five minutes duration. Longer periods of hypotension may cause margination of platelets in the blood vessels followed by the formation of a thrombus and embolism. Another result of long continued hypotension is damage to, and dilatation of, the capillary vessels. Still longer periods of hypotension may lead to the development of the lower nephron syndrome or to central necrosis of the liver. The time required to produce the conditions just enumerated is of considerable importance in assessing the safety of hypotension. Profound hypotension of several minutes duration may cause cardiac arrest and irreparable damage to the brain, while the liver and kidneys escape injury completely and would function without incident if the heart and brain could be restored to normal function. On the other hand, prolonged periods of moderate hypotension of, for example, 40 mm. of mercury mean pressure for a period of two or three hours, may not seriously damage the brain and heart, while causing conditions in the liver and kidneys which are not reversible. It is obvious, therefore, that both the duration of the hypotension and the level of the blood pressure must be considered. In making certain that controlled hypotension is to be safe, therefore, limitations must be put upon both the level and the duration of hypotension.

It is important to have an accurate means of reading blood pressure at low levels. In circumstances in which the blood pressure cuff and stethoscope fail, it is useful to have a manometer actually connected into the blood stream. A sterile mercury manometer can be used for this purpose, or an aneroid manometer, if kept upright and separated from the fluid by a volume of air, will read arterial pressures satisfactorily.

Speed infusion may be helpful in restoring failing blood pressure. The administration of blood or

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2. Griffiths, H. W. C., and Gillies, J.: Thoraco-Lumbar Splanchnicectomy and Sympathectomy: Anaesthetic Procedure, Anaesthesia **3**:134-146 Oct. 1948.

3. Enderby, G. E. H. and Pelmore, J. F., Controlled Hypotension and Postural Ischaemia to Reduce Bleeding in Surgery; Review of 250 Cases, The Lancet **1**:663-666, May 24, 1951.

4. Nicholson, M. J., Sarnoff, S. J., Crehan, J. P.: The Intravenous Use of A Thiophanium Derivative (Arfonad-RO 2-2222) For The Production of Flexible and Rapidly Reversible Hypotension During Surgery, Anesthesiology **14**:215-225, May 1953.

fluids may be accelerated by air pressure, by syringe, or by gravity. Blood may be administered rapidly through a large needle in an artery or vein by applying a measured air pressure to the flask containing the fluid. The danger of air embolism is great unless a safety dropper of some type is used which shuts off the flow of blood as soon as the flask is empty. Another satisfactory means of giving fluids rapidly is by the use of a 10 cc. ring syringe and either a three-way stop cock or a set of two valves. This method is particularly valuable in children in whom it may be desirable to give accurately measured quantities of blood at intervals. A third method of giving blood rapidly is by raising the vessel containing the fluid 6 or 8 feet above the patient, thus building up considerable pressure in the tubing. When any method of speed transfusion is used it may be important to warm the blood, as cold blood may cause spasm of the vessel and interference with a free flow. This can be accomplished readily by passing the blood through a length of plastic or rubber tubing which is laid in a basin containing water at a temperature of 100 degrees F.

HYPOTENSION BY SPINAL ANESTHESIA

Spinal anesthesia (particularly continuous spinal anesthesia) is well suited to providing hypotension. It is most appropriate when it can also supply the anesthesia in the field of surgery. Spinal anesthesia solution is injected through a catheter placed within the subarachnoid space and the use of 100 or 150 mg. of procaine ordinarily will give a satisfactory drop in blood pressure. This can be maintained by repeated injections as necessary.

ARTERIOTOMY HYPOTENSION

In a series of 50 cases of hypotension by arteriotomy the quantity of blood varied from 800 cc. to 3000 cc. The small quantity of blood was withdrawn from a woman of 106 pounds on whom a craniotomy was being performed. It is obvious that there is some loss of blood during a craniotomy and, in this case, this was added to that which was withdrawn from the radial artery. This may account for the satisfactory hypotension resulting from the relatively small withdrawal from the radial artery. The 3000 cc. was withdrawn from a man weighing 225 pounds undergoing operation for fenestration. In this operation the blood loss was very slight and, accordingly, a larger quantity of blood had to be withdrawn in order to provide hypotension.

The duration of hypotension is of great importance to the safety of the patient. The time in this series of 50 patients varied from zero (as soon as the blood had been withdrawn it was returned) to 130 minutes. The longest duration of the hypotension occurred in a patient having a fenestration, and during this time a considerable amount of his blood volume was made up by the passage of fluid from the tissue spaces into the blood stream, so that the blood pressure was not dangerously low during this long period.

The average data of 50 procedures is of considerable interest and is shown in the following table:

Average Data on 50 Procedures

(Arteriotomy Hypotension)

Amount of blood withdrawn cc.	Fall in Mean BP mm. Hg.	Time required for withdrawal minutes	Time that blood remained outside body minutes
1742.6	43.5	29.4	25.7

HYPOTENSION BY GANGLIOPLEGIA

Hexamethonium is the most popular drug for inducing hypotension by ganglioplegia. It is given in doses of 25 to 50 mg. and additional doses may be necessary. The blood pressure usually falls to a satisfactory level (70-80 mm. of mercury) in about five minutes, although some patients, particularly normotensive young adults, may fail to respond satisfactorily.

HYPOTENSION BY VASOPLEGIA

Arfonad acts on the ganglia, but perhaps even more on the musculature of the vessel wall itself. It is given intravenously in a solution (dextrose 5% in water) containing one milligram per cc. A prompt fall in blood pressure is produced, and can be maintained by a careful adjustment of the rate of flow. When the administration of the drug is discontinued the blood pressure usually returns to normal within five minutes.

COMBINED ARTERIOTOMY AND GANGLIOPLEGIA

It has been shown experimentally that the withdrawal of 10 per cent of a dog's blood will cause a drop in blood pressure which is restored, and over-compensated for, by the re-injection of this blood a few moments later. During the short period of hypotension there is a tendency for the blood pressure to return to normal from the point to which it dropped immediately upon the withdrawal of the blood. However, after the administration of hexamethonium, which causes a ganglionic block accompanied by vasodilatation, the withdrawal of 10 per cent of the animal's blood causes a more marked drop in blood pressure than before, and this is sustained without incident until the blood is returned to the animal. This procedure has been used clinically, as well as in the laboratory, and provides a highly controllable means of hypotension in circumstances during which it may be necessary to have a very low blood pressure for a very short period of time.

At the end of operation it is essential that the blood pressure be returned to normal before the incision is closed, so that all the bleeders can be properly controlled. This may be accomplished by the re-injection of the blood withdrawn in arteriotomy, or by the wearing off of, or counteracting of, hexamethonium or Arfonad by the administration of a vasopressor. Another method, which has been used on a few occasions, is the rapid arterial injection of 500 to 1000 cc. of saline, which gives a temporary rise in blood volume with an accompanying return of blood pressure to normal, during which the surgeon can control any bleeding points. Thereafter, the blood pressure falls again, as the saline rapidly leaves the vessels, and a slow return of blood pressure ensues.

CONCLUSION

Hypotension by different methods is a safe, a valuable, and, under some circumstances, even an essential procedure. The safety depends on close observation and control, and a safe blood pressure level maintained as short a time as possible.

The Negro as an Anesthetic Risk

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The apparent peculiar liability of the negro to sudden death under anesthesia has been noted by many observers. Sharp differences between mortality rates in white and negro patients have been recorded.[9] These observations suggest there are specific risks involved in the administration of anesthesia to these individuals. We wish to indicate some of the elements of this topic with which we are particularly familiar and to call attention to the pitfalls which may trap the unwary.

Diseases Affecting the Transport System

First, let us consider the diseases which affect the transport of physiologic gases and clinical conditions which increase vulnerability to hypoxia, a state which is potential in every anesthetic administration.

Cardiovascular Disease: The prevalence of cardiovascular disease is notorious, arteriosclerosis and hypertension being the main causes. Heart disease is of greater incidence than in the white race, occurs at a younger age, pursues a more rapid course and has a higher mortality rate. A hypersensitive vasomotor system renders these individuals especially liable to the development of essential hypertension. The course of vascular disease frequently is of the fulminating type, death occurring early from what often appears to be a combined insufficiency of heart, kidneys, and brain.[9]

Cardiologists agree that, in the presence of heart disease, the risk is increased in proportion to the extent with which the patient's activities are limited by his cardiac condition. This is a measure of his cardiac reserve, which is the important factor in evaluation. The ability of the heart to withstand the stress of a surgical operation depends upon whether or not this extra demand exceeds its reserve capacity.[8] If the cardiac patient has been free from symptoms suggesting myocardial insufficiency, functional capacity generally is adequate and anesthesia and operation are well tolerated. On the other hand, reserve is frequently inadequate and risk is increased in certain types of heart disease which are likely to cause sudden death.[6; 8]

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The two factors which may seriously embarrass the cardiac patient during operation are hypoxia and hypotension. Even a slight degree of hypoxia is poorly tolerated by the cardiac patient. Myocardial hypoxia, if long continued, will cause signs of myocardial insufficiency in patients who previously showed no signs of heart failure. In coronary heart disease the oxygen supply to the myocardium may be adequate at rest, but if hypoxia should occur during anesthesia, acute coronary insufficiency, with cardiac infarction or ventricular fibrillation, may ensue. Shock is more serious in individuals with cardiovascular disease than in normal patients. The circulatory system has lost much of its compensatory mechanism through arteriosclerosis and degeneration. In the presence of hypertension and sclerotic changes in the peripheral vessels, a moderate degree of peripheral vascular collapse may result in serious impairment of the functioning of vital organs.[6; 8; 10]

No generalization can be made regarding the choice of anesthesia for the cardiac patient. All methods have been condemned at one time or other. There is agreement on one point: the avoidance of hypoxia of any type is of such importance that it allows for no modification.[6]

Sickle Cell Disease: That sicklemia at times is responsible for otherwise unexplained deaths following anesthesia and other states which produce a lowering of oxygen tension is now recognized.[12]

Sickle cell disease is a chronic disease varying in severity and in age of onset. Figures for the American Negro approach an incidence of eleven per cent. Within the group who inherit the sickling trait, approximately one in forty has sickle cell anemia. Findings indicate that sickling depends upon an hereditary factor, the presence of a sufficient quantity of abnormal hemoglobin within the erythrocyte. One of the characteristics of the abnormal hemoglobin found in sickle cell disease is that it crystallizes on deoxygenation and goes back into a solution on oxygenation. The crystals forming within the red cells distort the cell membrane and cause the cells to assume multipointed shapes called "sickle cells." These forms produce "log-jams" in the blood vessels and cause circulatory stasis. Stasis in turn favors hypoxia, which exaggerates the sickling process, thus producing a vicious cycle.[12]

Patients with sickle cell anemia usually have manifestations of the disease in infancy. There is chronic anemia and jaundice throughout life. These people are subject to recurrent febrile and painful illnesses called "crises" which are best explained by obliterative vascular lesions. Death usually occurs during childhood or in the early adult years. As a rule, patients with the sickle cell trait have no symptoms or clinical signs related to their inherited abnormality. However, under hypoxic conditions, manifestations of the disease will develop.[12]

The red cells are extremely vulnerable to hypoxia, a condition which is potential in every anesthetic administration. Low oxygen tension may be produced by increased metabolism, as occurs in fever or exercise. Congestive heart failure, mild transfusion reactions, high altitude flying, and peripheral circulatory failure induce hypoxic states and accelerate the sickling phenomena. Individuals who have suffered from various types of trauma which have produced shock and who eventually undergo general anesthesia after recovering from shock, often succumb for reasons that are altogether vague from clinical observation. Profound sickling will be the only pertinent finding at post mortem examination.[12]

That there is no anesthetic method without hazard for sicklemic patients is evidenced by the fact that all types have been listed as contraindicated. This would seem to preclude anything but faultless conduct of the most suitable anesthetic procedure.[12]

RESPIRATORY INSUFFICIENCY

Now let us consider the technical aspects of handling these patients, who are peculiarly vulnerable to hypoxic states.

SIGNS OF HYPOXIA AND HYPERCARBIA: In the average patient who is not under the influence of depressant drugs, the clinical signs of asphyxia may be easily recognized. In the anesthetized patient, the general signs and symptoms of oxygen want and of carbon dioxide excess are obscured. The warning signs of hypercarbia and of hypoxia vary with the depth of anesthesia and with each drug employed. Death from acute asphyxia may occur without the intervention of such significant developments as a slow bounding pulse, a gradual arterial pressure fall or a period of asphyxial gasping.[2; 3]

These manifestations depend upon the response of the vagi, the cardio-accelerators, the vasoconstrictors, the carotid and aortic bodies, the carotid sinus and many other components, including the hypothalamus and the adrenal glands. Most of the anesthetic agents commonly employed probably depress chemoreceptor response in the lighter planes of surgical anesthesia. When these drugs are employed to produce deeper planes, the medullary centers are so depressed that it is unlikely that any blood pressure or pulse variations would appear coincident with severe oxygen want.[2] The effects of preanesthetic medication are not generally appreciated. Certain of these drugs further interfere with the clinical signs of hypoxia.[3]

The recognition of carbon dioxide retention is especially difficult in the presence of depressant drugs. This may be due to the fact that carbon dioxide in many ways acts as an anesthetic agent. The development of its effects often is insidious. Moreover, these effects merge imperceptibly with the effects of narcosis and may be masked entirely by the use of depressant drugs.[7]

One cannot overemphasize the fact that in dark-skinned patients cyanosis may not be readily detected. The many shades of black and the pigmentation of mucous membranes and nail beds add to the difficulty of judging changes in color. It should be noted that pupillary changes may be obscured by the heavy pigmentation of the iris.[2]

These facts may help to explain why even experienced anesthetists have allowed irreversible damage to develop without recognizing the period of distress.[2]

HYPOVENTILATION: The anesthetist is intimately concerned with ventilation because it is essential to the patient's welfare to insure respiratory sufficiency.[7] Hypoventilation is that state in which there is inadequacy in the exchange of gas between ambient atmosphere and blood because of alterations in pressure differentials, volume of gas moved or changes in the time during which gas is moved. In other words, ventilatory insufficiency (hypoventilation) results in respiratory insufficiency. Hypoventilation interferes with the transport of oxygen, carbon dioxide and anesthetic gases and vapors. This condition leads to hypoxia, hypercarbia and inadequate or excessive anesthesia. In addition, hypoxia causes excessive mucous secretions which further interfere with gaseous exchange in the lungs.[7]

It is especially important to remember that hypoxia can be compensated for by increasing the tension of oxygen in the respired atmosphere, but hypoventilation and hypercarbia still may exist. It must be reiterated that the patient may be in a state of severe hypoventilation without showing clinically detectable signs of hypoxia and/or hypercarbia.[7]

A potent source of hypoventilation is obstruction of the airway, the most common and pernicious complication of general anesthesia.[1] A valve-type obstruction of the upper airway, due to an overhanging epiglottis, is said to occur very rarely. That it does occur we have demonstrated to our complete satisfaction on several occasions. The mechanism of the obstruction consists of four factors: 1. a soft flexible epiglottis, 2. muscular relaxation during general anesthesia, 3. force of gravity when the patient is lying on his back, 4. air velocity through the larynx.

Cessation of respiratory exchange is the only sign of trouble. No sound is produced. A silent obstruction is characteristic of the impacted epiglottis. The procedure for relief of the obstruction consists of the following maneuvers:

1. Clearing the pharynx of foreign material.
2. Freeing the epiglottis
    - A. Put traction on the tongue; forcing the base of the tongue forward draws the epiglottis up and frees it.
    - B. Digital manipulation of the larynx can relieve the impaction. The epiglottis is lifted from the side with the finger.
3. Artificial respiration with oxygen.[4; 5; 11]

A catheter passed translaryngeally into the trachea and kept free from kinks and with a clear lumen is a guarantee against recurrence of obstruction.

SUMMARY

To recapitulate, hypoxia is a factor of grave importance. The negro patient is peculiarly susceptible to hypoxia due to the prevalence of circulatory diseases and to the presence of certain physical factors affecting the conduct of anesthesia.

Anesthetists are commonly using drugs and technics which tend to produce one or more forms of hypoxia. It may not be amiss to mention the possibilities of harm in heavy premedication, hypotension, localized stasis from surgical posture, and respiratory depression inherent in the nature of certain anesthetic agents. Those patients exhibiting relative hypoxia prior to operation, no matter what the cause, are operative risks. This is because the hypoxia already existent is made worse by addition of other types of hypoxia through use of preoperative medication and anesthesia, and through shock due to various causes. The general signs and symptoms of hypoxia and hypercarbia are obscured by anesthesia. The use of certain premedicant drugs in doses which may mask the signs of asphyxia are dangerous. In dark-skinned patients cyanosis may not be readily detected and pupillary changes may be obscured by heavy pigmentation of the iris. One must be aware of the possibilities to foresee and forestall them.

The gravest danger to the patient is the possibility of poor respiratory exchange. Many of the fatal complications produce damage by interfering with transport of oxygen and carbon dioxide in the body. Expert application according to the patients' needs is *sine qua non* of safe and satisfactory anesthesia.

REFERENCES

1. Adriani, J.: Techniques and Procedures of Anesthesia. Charles C. Thomas, Springfield, 1947, pp. 139-146.
2. Allen, C. R., Echols, R. S., Hoeflich, E. A., O'Neal, K. C., and Slocum, H. C.: Variations in the Signs of Acute Oxygen-Want during Anesthesia, Anesthesiology **8**: 601-614, 1947.
3. Allen, C. R., Echols, R. S., O'Neal, K. C., and Slocum, H. C.: The Effects of Premedication Upon the Signs of Asphyxia during Nitrous Oxide-Oxygen Anesthesia, Anesthesiology **10**: 164-174, 1949.
4. Caiger, G. H.: The Role of the Epiglottis in Anesthetic Deaths, J. Laryngology and Otology **57**: 250-263, 1942.
5. Caiger, G. H., and Sichel, D. A. S.: Obstruction of the Larynx During Anaesthesia, Anaesthesia **9**: 177-184, 1954.
6. Collins, V. J.: Principles and Practice of Anesthesiology, Lea and Febiger, Philadelphia, 1952, Chapter 58.
7. Cullen, S. C.: Anesthesia in General Practice, 4th Edition, Year Book Publishers, Chicago, 1954, Chapters 2 and 3.
8. Mason, R. L., and Zintel, H. A.: Preoperative and Postoperative Treatment, 2nd Edition, W. B. Saunders Company, Philadelphia, 1946, Chapter 5.
9. Poe, M. F.: The Negro as an Anesthetic Risk, Anesthesiology, **14**: 85-89, 1953.
10. Poe, M. F., and Karp, M.: Anesthesia in Patients with Cardiovascular Disease, Unpublished Data.
11. Pokrzywnicki, S.: Impaction of the Epiglottis during Anesthesia, Anaesthesia, **8**: 47-48, 1953.
12. Shapiro, N. D., and Poe, M. F.: Sickle Cell Disease: An Anesthesiological Problem, Anesthesiology, In Press.

The Shorter Acting Muscle Relaxants

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The clinical introduction of Curare as a muscle relaxant by Griffith[1] in 1942, opened the gates to investigators and the stampede was on to produce newer and better natural and synthetic agents which were capable of effecting muscle relaxation. Some of these agents have proved to be less desirable than the original Curare, while others are definite improvements. At any rate, the introduction of muscle relaxants appears to be a significant development in Anesthesia.

Originally all drugs which produced muscle relaxation were known as "Curare-like" drugs. It is now known, however, that one may produce muscle relaxation by an entirely different method than that used by Curare. Consequently, we now speak of this group of drugs as "muscle relaxants" rather than "Curare-like," although Curare still holds its position as the drug for standard comparison.

In order to understand the mode of action of the muscle relaxants, it is well that we review briefly one theory of neuromuscular transmission.[2]

If we consider a single motor nerve and muscle in the body, we note that they both have the property of transmitting a wave of excitation which eventually ends in the contraction of the muscle fiber. This action in both the nerve and muscle fiber is dependent on the fact that the surface membranes are electrically charged. At rest, the outside of the membrane has a positive charge with a negative charge being inside the membrane. Stimulation of the nerve results in a reduction in the membrane potential with the outside of the membrane now becoming negatively charged and the inside of the membrane positively charged. The membrane is then said to have been activated or "depolarized". This wave of excitation continues down the nerve fiber with one area depolarizing the area immediately ahead of it until the nerve ending is reached. The depolarization is transient in nature, rapidly returning to its resting state ready to transmit another impulse. The nerve filaments and the muscle tissue make extensive contact with one another, but there is no actual continuity of tissue. A structure known as the "endplate" has recently received a great deal of

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attention, and this structure appears to fill the anatomical gap between the nerve endings and the muscle fibers. The exact anatomy of the endplate is not clearly understood but it appears that the impulse from the nerve results in the production of acetylcholine, which serves as a chemical transmitter and "depolarizes" the endplate, thereby producing a potential known as the "endplate potential". When this potential reaches a certain critical level it "depolarizes" the membrane of the muscle fiber, setting up a muscle action potential with a muscle contraction resulting.[3]

Cholinesterase is an enzyme which is also found in high concentration at the motor endplate. This enzyme rapidly destroys acetylcholine to insure that the acetylcholine normally released at each nerve impulse gives rise to only a single muscle response and does not persist in its action.

Bearing in mind the forementioned theory of neuromuscular transmission, it is possible to account for four main methods of producing neuromuscular block and thereby muscle relaxation.[4]

A. "Competitive" Block. — If one introduces a substance into the body which has an affinity for the receptors at the endplate similar to that of Acetylcholine, one could lessen the effectiveness of Acetylcholine through the competition for receptor site and thereby render a given quantity of Acetylcholine less active. The endplate potential would then not be sufficient to result in a propagation of the wave across the bridge from the nerve to the muscle fiber and the muscle would not contract.

In order to counteract this block, one may introduce a drug which inhibits the action of Cholinesterase — an Anticholinesterase—such as Tensilon or Neostigmine. By blocking the action of Cholinesterase, which normally destroys Acetylcholine, the level of Acetylcholine is allowed to build up more quickly to the critical level necessary to effect depolarization.

B. Depolarization Block. — This type of block is that which is produced when one administers a drug that has an action similar to Acetycholine but which is not destroyed by Cholinesterase. A drug of this type would produce depolarization at the endplate with propagation of the impulses into the muscle fibers with contractions resulting. However, since the drug is not hydrolyzed at the neuromuscular junction, the depolarization persists and it is impossible to produce another muscular contraction until the endplate and muscle fiber have been allowed to repolarize and return to the state of rest.

If one introduces an Anticholinesterase drug in the presence of this type of block, the block is potentiated rather than antagonized since further Acetylcholine action serves to prolong the depolarization. Hence, Tensilon and Neostigmine are contraindicated in the "depolarization" block.

C. Anti-acetylcholine Production Block.—This type of neuromuscular block could be brought about through the administration of a drug which would prevent the production of Acetylcholine at the myoneural junction, thereby eliminating the chemical necessary for the transmission of the impulse from the nerve to the muscle. This type of block, at the moment, is only of theoretical importance, since the drugs now known to produce this type of block are hardly of practical importance. Botulinum Toxin and Procaine supposedly produce their block in this fashion, but the block produced by the former agent is irreversible, and that produced by the latter is in the toxic range.

D. Mixed Type Block.—The fourth type of block which can be produced is that resulting through the administration of a drug which does not act in a pure form but rather through a combination of A and B. This type of drug acts in a competitive fashion as well as depolarizing. Ordinarily the administration of an anticholinesterase as an antidote for this type of drug will diminish, but will not completely abolish, its blocking characteristics.

For this discussion, four separate muscle relaxants have been chosen: Gallamine Triethiodide (Flaxedil), Decamethonium Bromide (Syncurine), Benzoquinonium Chloride (Mytolon), and Succinylcholine.

FLAXEDIL

Flaxedil is a synthetic muscle relaxant which acts at the myoneural junction in a competitive blocking manner similar to that of Curare.[5] This drug appears to be more predictable on a dose governed by the patient's weight than is Curare. The usually recommended initial dose is approximately 60 to 80 milligrams or 1.0 to 1.5 milligrams per kilogram of body weight.[6] The onset of action of this drug is usually heralded by a tachycardia to about 1/3 more than the patient's normal heart rate. The tachycardia appears to be on the basis of an inhibiting action on the cardiac vagus and this action persists long after the relaxing effect of the drug is over. There appears to be no significant clinical effect on the blood pressure or cardiac rhythm. The peak of action occurs rapidly in 4 to 6 minutes with the duration of clinical relaxation being rather short, and estimated at 10 to 15 minutes. If one measures the respiratory volume, however, a depressed effect can be noted for as long as 40 minutes. It is important to realize that there is a marked additive action on successive doses so that one must cut the size of each repeated dose to obtain the desired amount of muscle relaxation. Flaxedil does not cause bronchospasm and appears to possess no histamine-like action. Flaxedil also differs from Curare in that one does not observe an appreciable difference in effect when used in equal doses with Ether or Cyclopropane. This drug is excreted from the body by the kidneys and approximately 70 percent of the administered dose can be recovered unchanged in the urine.[7] As one could predict from the competitive type block which it produces, Flaxedil is effectively antagonized by Tensilon and Neostigmine.

SYNCURINE

Syncurine is likewise a synthetic muscle relaxant possessing two quaternary nitrogen groups separated from one another by ten Carbon atoms hence often called C-10. This drug differs in its method of production of muscle relaxation from that of Flaxedil in that it acts through persistent depolarization as described in B. Syncurine has an extremely rapid onset of action with a short duration of clinical relaxation of approximately 5 to 15 minutes. The optimum dosage varies from patient to patient with an average of 2 to 3 milligrams of Syncurine being given as an initial intravenous dose. Syncurine possesses no additive or cumulative effect in contrast to most of the other muscle relaxants; in fact if one gives a second and equal dose of this agent thirty minutes after the initial administration, there is a failure to bring about an equal decrease in muscle tone, thereby demonstrating tachyphylaxis to the effects of Syncurine on the striated muscles.[4] Syncurine possesses an extremely narrow margin between adequate abdominal muscle relaxation and apnea. It does not produce major circulatory changes nor does it liberate histamine.[8] This drug is excreted from the body primarily through the kidneys. Inasmuch as this drug produces its relaxing effect through "depolarization", it is not antagonized or inhibited by anticholinesterases, such as Tensilon or Neostigmine. It is of interest to note that the action of Syncurine can be prevented by a preceding dose of Tubocurarine.

MYTOLON CHLORIDE

Mytolon is a synthetic muscle relaxant which has a somewhat slower onset of action than the previously discussed agent although it, too, has a similar duration of clinical action of approximately 10 to 15 minutes. Milligram for milligram, the potency of this drug is approximately equivalant to that of D-Tubocurarine with the average initial injection ranging from 9 to 15 milligrams. Repeated injections of this drug should be reduced to approximately 1/2 the initial dose so as not to produce a greater activity than one desires. Mytolon has one very distinct disadvantage and that is that in the doses used in clinical anesthesia, the drug serves as a stimulant to the parasympathetic ganglia. This stimulation results in excessive salivation requiring repeated large doses of Atropine or Scopolamine to keep the secretions under control.[9] The mechanism of action of Mytolon is not entirely clear. It appears to be a mixed type action combining the "competitive" block and the depolarization block. Mytolon resembles Curare more than Syncurine in its mechanism of action. It is potentiated by Ether and it is antagonized to some extent by the anticholinesterase drugs such as Tensilon or Neostigmine.[10] Mytolon is excreted by the kidneys with approximately 75 to 80 percent being recovered unchanged in the urine.[11]

SUCCINYLCHOLINE CHLORIDE

Succinylcholine is an ultrashort acting relaxant drug. Its peak of action occurs in one or two minutes with a duration of clinical relaxation normally occuring for a period of four to five minutes following a single relaxing dose.[12] When it is desirable to produce muscle relaxation for short periods of time, such as for intubation and manipulative procedures, the drug is usually administered in a dosage of 20 to 40 milligrams.[13] Because of its ultra-short action, Succinylcholine is equally effective in producing long term relaxation when administered as a 0.1% solution by continuous drip. When administered by the continuous method, one is able to titrate the degree of muscle relaxation which is desired and one thereby avoids marked overdosage with its accompanied prolonged apnea. One often sees muscle fasciculations following the injection of Succinylcholine, but these appear to be of little clinical significance if the patient is asleep when they occur. The occurrence of muscle fasciculations can be decreased if the rate of intravenous administration is slow. A slight increase in pulse rate is a common accompaniment of the use of this drug. Succinylcholine differs from the other muscle relaxants in that it is hydrolyzed by Plasma Pseudocholinesterase.[14, 15]

There are two known types of enzymes which are capable of hydrolyzing Acetylcholine in the body. One is the so-called "true Cholinesterase," which is located predominantly at the myoneural junction. It is responsible for hydrolyzing the Acetylcholine formed at this point. The second enzyme, the so-called "Pseudocholinesterase" or "Plasmacholinesterase," is found primarily in the blood stream. It is more nonspecific in that it will hydrolyze a number of ester type compounds. It is this latter enzyme which is responsible for the breakdown of Succinylcholine as well as some of the local anesthetic agents such as Procaine.

Plasma Cholinesterase is diminished in patients with low plasmaprotein levels such as that seen in patients suffering from liver disease, malnutrition, or severe anemias. Also individuals who have had excessive contact with anticholinesterase insecticides or certain specific war gasses may also exhibit diminished plasmacholinesterase activity. A number of cases with prolonged apnea following the use of Succinylcholine have been reported in the literature, and most of these individuals have been shown to have a low circulating Plasmacholinesterase. It has been suggested also, that patients who sustain definite intestinal trauma may exhibit a cumulative effect of Succinylcholine with prolonged apnea.[16]

DISCUSSION

It is now 12 years since Dr. Harold Griffith first introduced a muscle relaxant into clinical anesthesia. The need for such a group of drugs is obvious as one observes the rapid acceptance of each new agent by anesthetists throughout the world. Reviewing the history of the introduction of each new agent, one notes the general pattern of, first, extreme enthusiasm followed later by lassitude as more and more reports of toxicity creep into the literature. At the moment, the last drug discussed, Succinylcholine, is receiving the greatest support, and in our hands we find this to be the most successful of all the muscle relaxants to date. However, even this drug, seemingly to us the best in the group, is far from ideal. The tendency is to think of each drug purely as a muscle relaxant instead of considering its effect on the body as a whole. A recent review of the deaths associated with anesthesia from ten reasonably comparable departments of anesthesia reveals that the mere introduction of muscle relaxants into general anesthesia raises the death rate to five times that of the incidence of death in general anesthesia in the non-muscle relaxant group.[17] This is a telling blow to even the most conservative anesthetists. The fact that a common cause of death from muscle relaxants was due to circulatory collapse in this report leads one to think that possibly many of the deaths were in cases that had received "Curare", which has a markedly greater depressant effect on the circulation, plus a tendency to histamine release which the synthetic muscle relaxants do not exhibit. At any rate, it would be interesting to see a comparison of deaths in patients receiving muscle relaxants broken down percentage-wise for each specific agent.

Since the muscle relaxant group fulfills a real need in the operating room, it would seem wise to take a conservative attitude toward the clinical use of this group of drugs. It is our opinion that these drugs should be used with specific indications and that they should not be employed routinely when other proven methods are quite adequate. No drug is ever foolproof and the safety in its use rests primarily with the individual at the head of the table. If the individual anesthetist is not able to cope with the "unusual" reactions to the drug, it makes little difference which muscle relaxant is used. In the hands of the trained anesthetist, it would seem to us that Succinylcholine is doubtless an improvement over Curare as a muscle relaxant. We believe that some muscle relaxant will eventually hold a permanent place as an adjunct to anesthesia, but complete evaluation cannot be made at this time for want of adequate studies.

SUMMARY

1. Stimulation of a motor nerve results in the production of Acetylcholine at the nerve endings. The Acetylcholine serves as a chemical transmittor and "depolarizes" a structure known as the endplate. When the endplate potential reaches a given critical level, it "depolarizes" the muscle fiber, thereby producing a muscle contraction. Another enzyme found in high concentration at the myoneural junction is Cholinesterase. This enzyme serves to destroy Acetylcholine and prevents a persistent transmission of nerve impulse.

2. Four possible methods of producing neuromuscular block are discussed. They are: (a) "Competitive" Block — produced by the injection of a substance which unites with the receptors at the endplate, preventing Acetylcholine from transmitting the

impulse to the endplate. D-Tubocurarine is an example of a muscle relaxant acting in this fashion; (b) Depolarization Block—produced by a drug having a similar action to Acetylcholine, but one which is not destroyed by Cholinesterase. A drug of this type produces continuous depolarization at the endplate without permitting repolarization; (c) Prevention of Acetylcholine Production—a method of producing a block which at the moment is of only theoretical importance. This could be brought about through the administration of a drug that prevents the production of Acetylcholine at the myoneural junction, and therefore prevents the chemical transmission of the impulse from the nerve ending to the endplate; (d) Mixed Type Block—a type of block produced by drugs acting in any combination of A, B, or C.

3. Flaxedil is a muscle relaxant which has a duration of clinical relaxation of approximately 10 to 15 minutes. It has an additive effect and one must decrease the size of successive doses to obtain the desired muscle relaxation. The drug produces a tachycardia. It acts in a competitive type block, as does Curare, and is effectively antagonized by Tensilon and Neostigmine.

4. Syncurine produces a duration of clinical relaxation of approximately 5 to 15 minutes. Tachyphylaxis accompanies repeated administrations of this drug. It acts through "depolarization" and consequently the anticholinesterase drugs are not effective antidotes.

5. Mytolon produces clinical muscle relaxation for a period of 10 to 15 minutes. It has the great disadvantage of stimulating the parasympathetic ganglia resulting in excessive salivation. This drug acts by combining the "Competitive" Block and the "Depolarization" Block.

6. Succinylcholine is an ultrashort acting drug producing muscle relaxation for a period of 4 to 5 minutes following single dose administration. When administered as a 0.1% solution, the degree of muscle relaxation desired can be titrated for as long a period as the surgery requires. Succinylcholine acts through "depolarization". In contrast to the other muscle relaxants, this drug is destroyed by the circulating plasma pseudocholinesterase.

REFERENCES

1. Griffith, H. and Johnson, G.: The use of curare in general anesthesia. Anesthesiology **3**:418-20, July 1942.

2. Wright: Applied physiology. 9th Edition, 510.

3. Paton, W.D.M.: The principle of neuromuscular block anesthesia **8**:151-174, July 1953.

4. Paton, W.D.M.: The pharmacology of decamethonium Ann. New York Acad. Sc. **54**: 347-361, October 1951.

5. Ricker, W.F., and Wescoe, W.C.: The pharmacology of flaxedil with observation on certain analogs. Ann. New York Acad. Sc. **54**:373-394, October 1951.

6. Artusio, J.F., Jr.: Rucker, W.F. Jr.: and Wesco, W.C.: Studies on inter-relationships of certain cholinergic compounds; anticurare action in anesthetized man. J. Pharmacol. and Exper. Therap. **100**:227-257, October 1950.

7. Musin, W.W.; Weir, R.; Mason, D.F.J.; and Langston, G.T.: Curare-like actions of tri - (diethylaminoethoxy) — benzine triethyliodide (R.P. 3697, Flaxedil) Lancet **1**:726, April 30, 1949.

8. Grob, D.; Holaday, D.A.; and Harvey, A.Mcg.: Effects of bistrimethylammonium decane dioxide and dibromides on neuromuscular function and on induced convulsions in man. New England J. Med. **241**:812-815, November 24, 1949.

9. Arrowood, J.G.: Mytolon chloride—new agent for producing muscular relaxation; Preliminary report. Anesthesiology **12**:753-761, November 1951.

10 Foldes, F.F.: The use of mytolon chloride in anesthesiology. Ann. New York Acad. Sc. **54**:503-509, October 1951.

(Continued on page 293)

Prevention and Treatment of Cardiac Emergencies During Anesthesia

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The topic for discussion is one of utmost importance. The literature today is filled with reports of cardiac arrest: its causes, prevention and treatment. The surgical team no longer includes the kitchen table and drip ether anesthesia with the surgeon either alone or, if fortunate, with one assistant. However, in those "good old days" cardiac arrest was reported to be 1-10,000 cases. Now with our "advances" it occurs in 1-5000 cases. The surgical team today includes the surgeons and assistants, numerous surgical and circulating nurses, the anesthetist and anesthesiologist, as well as the internist whose primary interest is in cardio-respiratory disorders and function. Experience in our hospital surgery has led us to use first the direct writing electrocardiograph and later the combination Cambridge oscilloscope electrocardiograph as standard operating room equipment. Since anoxia is considered to be the most important factor in arrhythmias occurring during surgery, we have added an oximeter to our equipment.

Every case for surgery should be studied carefully, analyzing the cardio-respiratory system accurately. If time permits, every case should have a chest film. Every major surgical case over 30 years of age or any with a cardiac abnormality should have an electrocardiogram. Anesthetic difficulties frequently occur when these studies are omitted. A careful history is most important. This includes possible experience with rheumatic fever, streptococcal infections and diphtheria. The patient's tolerance to exercise as regards dyspnea, anginal pain, cough and fatigability is important to know. The examination of the heart determines its size and murmurs and their significance. If disease exists, it should be carefully classified as to etiology, anatomical defect, physiological status and functional and therapeutic classifications. If such a survey is made, cases of incipient congestive failure, arrhythmias, anemia, beriberi and other nutritional and electrolyte defects and respiratory abnormalities will be detected and can be corrected preoperatively. The anesthetist should become acquainted with the patient before putting him to sleep. These attentions will put the patient in better psychic balance.

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The arrhythmias are the greatest problem during anesthesia. These may occur in normal hearts, but more frequently in abnormal hearts. We studied the age and incidence of arrhythmias according to cardiac status.[1] (See Table I) There were 30 cases (47 per cent) in the normal group with ages ranging from 23-73 (average 52) who did not have an arrhythmia. In the normal group of cases there were 34 (53 per cent) who sustained an arrhythmia. The age range was essentially the same (22-75 with average age 50). There were 9 cases (26.4 per cent) who had no abnormality in rhythm in the abnormal heart group. Ages ranged from 57-73 with an average of 65. However, 25 cases (73.6 per cent) in this group developed an arrhythmia. Their ages ranged from 43-91 with an average of 62. Four cases (26.7 per cent) classified as possible heart disease had no arrhythmia. Ages ranged from 46-77, with an average of 61. Eleven cases (73.3 per cent) so classified developed an arrhythmia. The age range was 37-76 with an average of 55. The main thing to note here is the high incidence of cardiac arrhythmias with normal heart findings; however, arrhythmias were definitely more common in abnormal hearts.

TABLE I

Heart Condition	No Arrhythmia	Arrhythmia
Normal	30 cases (47%) Age range 23-73 Average age 52	34 cases (53%) Age range 22-75 Average age 50
Abnormal	9 cases (26.4%) Age range 56-73 Average age 65	25 cases (73.6%) Age range 43-91 Average age 62
Possible heart disease	4 cases (26.7%) Age range 46-77 Average age 61	11 cases (73.3%) Age range 37-76 Average age 55

Classification of Causes of Cardiac Arrhythmias and Arrest

1. Type of anesthetic agent and over-anesthetization.
2. Hypoxia or anoxia.
3. Hypercapnia.
4. Hypotension and shock.
5. Over-medication before and during anesthesia.
6. Nutrition, anemia, and electrolyte balance.
7. Cardio-respiratory and vascular status.
8. Psychosomatic status.
9. Type and duration of operation.
10. Reflexes from the surgical field — the so-called vagovagal reflex.

The main thing we wish to stress is the type of anesthetic agent. Any anesthetic agent is unsafe if improperly administered. All are myocardial depressants and trouble has occurred with all types. Direct stimulation of the vagus nerve in the dog will not produce cardiac arrest in the absence of hypoxia. In humans, the passage of an intratracheal tube does not cause arrhythmias unless the patient is hypoxic.[2] This suggests that hypoxia is the factor in humans when the so-called vago-vagal field reflexes are mediated.

We studied the incidence of cardiac arrhythmias during anesthesia comparing cyclopropane and pentothal-nitrous oxide-ether anesthesia.[1] We routinely made electrocardiograms on patients undergoing major surgery. Taylor[3] in 1941 reported an incidence of only 6.5 per cent arrhythmias in 41,690 patients under cyclopropane anesthesia, whereas we and others, with the aid of the electrocardiogram, have found a much higher percentage of arrhythmias, especially extrasystoles of ventricular origin.

Table 2 lists 114 arrhythmias encountered in 113 procedures in which cyclopropane was used. One hundred five patients were given only cyclopropane in conjunction with the routine preoperative medication of 10 mg. to 15 mg. (1/6 to 1/4 grain) morphine and 0.4 mg. (1/150 grain) atropine. Four patients were given a pentothal induction followed with cyclopropane and then shifted to ether.

TABLE II

Type of Rhythm		Frequency
Ventricular rhythms		85
Multiple and multifocal ventricular premature contractions	41	
Pulsus bigeminus	23	
Occasional ventricular premature contractions	15	
Ventricular tachycardia	3	
Gross dysrhythmia	2	
Ventricular fibrillation and cardiac arrest	1	
Auricular rhythms		18
Auricular premature contractions	11	
Wandering pacemaker	5	
Auricular fibrillation	2	
Supraventricular Tachycardia		2
Nodal Rhythms		9
Shift in pacemaker from sinus to A-V node	2	
Nodal premature contractions	6	
Nodal rhythm	1	

Forty-three (38 per cent) of the 113 patients had no disturbance in rhythm. One of these had 0.1 per cent procaine dripping intravenously from induction until the end of surgery. Seventy patients (62 per cent) had some type of arrhythmia as listed. Cases of sinus tachycardia were usually transient and not included. The multiple and multifocal ventricular premature contractions were the most frequent arrhythmias and are generally considered the most dangerous. The heart may go into a gross dysrhythmia and ventricular fibrillation from this stage as well as from ventricular tachycardia. Cardiac arrest developed in one case in this group.

Table 3 summarizes the incidence of arrhythmias in 221 patients under pentothal induced, nitrous oxide - ether anesthesia. Twenty-one patients showed some type of arrhythmia. Eight patients had one or more types of ventricular rhythm. Three patients had nodal rhythms; eleven had one or more auricular rhythms. In this group there was also one case of cardiac arrest.

The agent of choice for counteracting these arrhythmias in this study was intravenous procaine. Carter and Eisaman[4] reported on the powerful depressant action of intravenous procaine on the myocardium and conduction system. This depressant action has been found beneficial in ventricular arrhythmias. Hearts suffering from inflammatory or degenerative lesions may tolerate only a fraction of usual dosages; therefore, control electrocardiograms should always be made. The speed of injection of intravenous procaine may influence its toxic effect upon the heart. For this reason we feel that rather than direct intravenous injection of 100 mg. of procaine it is better to use a continuous drip.

In most cases of major surgery fluid is being injected intravenously for multiple purposes. In this glucose or saline solution 5 cc. of 20 per cent procaine are instilled when indicated, making a 0.1 or 0.2 per cent solution of procaine, depending upon whether it is 1,000 or 500 cc. initial volume. The rate of drip can be

TABLE III

Ventricular rhythms (8 cases)		14
Multiple and multifocal ventricular premature contractions	4	
Occasional ventricular premature contractions	6	
Paroxysmal right bundle branch block	1	
Ventricular tachycardia	1	
Cardiac arrest	1	
Pulsus bigeminus	1	
Auricular rhythms		19
Wandering pacemaker	12	
Prolonged P-R interval	4	
Auricular premature contraction	2	
Auricular fibrillation	1	
Nodal rhythm		3

TABLE IV

Type of Rhythm	Restored				Not Restored		Normal	
	Ether and Procaine	Shift to Ether	Procaine Drip	Spontaneously	Cardiac Arrest	Procaine Not Given	Nothing Added	Continuous Procaine Drip
Normal rhythm							42	1
Ventricular arrhythmias	1	1	18	14	1	9		
Combined supraventricular and ventricular arrhythmias			8	9		2		
Various auricular and nodal arrhythmias						7		
Totals	1	1	26	23	1	18	42	1

adjusted as to the need. At first it may flow in freely; when the rhythm is improved or controlled, the rate may be established at 80-100 drops per minute. This is a safe procedure in practically all patients able to undergo surgery. We believe that the direct intravenous use of procaine along with that of quinidine, intravenously, may well be very dangerous. They seem to have a synergistic effect in producing prolonged QRS conduction time as well as other depressant effects.

Table 4 gives our results[1] with the treatment of arrhythmias occurring with cyclopropane anesthesia. Twenty-nine patients (25.6 per cent) had procaine, intravenously. Only one patient had it from beginning to end of surgery; arrhythmia did not develop in this case. Eighteen patients with ventricular arrhythmias had the rhythm restored to normal with intravenous procaine (3 patients were greatly improved but not completely restored to normal). One patient required procaine plus a shift to ether to restore normal rhythm and one returned to normal with ether. Fourteen cases of ventricular arrhythmias were restored to normal without therapy. Procaine was not given in nine other cases of ventricular arrhythmias but the rhythm was not restored to normal. Intravenous procaine was used only in the more alarming situations. This included twenty (45 per cent of the group) of the ventricular arrhythmia group. One case required a shift to ether and cardiac arrest developed in another case. Thus, in this group intravenous procaine might be considered 90 per cent effective.

In the combined supraventricular and ventricular arrhythmias the rhythm was restored to normal in nine cases without therapy other than increased oxygen supply. However, in eight cases it was believed necessary to give intravenous procaine and in these cases all were restored to normal (one patient had an occasional auricular premature contraction and in another the chronic auricular fibrillation was not influenced).

In the other cases of simple auricular arrhythmia and the case of supraventricular tachycardia intravenous procaine was not used. Thus, in this series of twenty - nine cases, intravenous procaine might be considered to be 93 per cent effective in restoring rhythm.

Intravenous procaine was used in only five instances in the ether group (2.2 per cent). In the three cases in which it was used for multiple and multifocal ventricular premature contractions it was successful in restoring rhythm to normal. In a case of cardiac arrest it did not restore the rhythm to normal. The rhythms varied between multiple and multifocal ventricular premature contractions, periods of ventricular rhythms and tachycardia and arrest periods lasting as long as nine seconds before complete asystole occurred. Figure I illustrates the beneficial results of intravenous procaine in combating multiple and multifocal ventricular premature contractions. One, two and three electrocardiograms in the left hand column represent the standard limb leads taken before induction. The lead (2) at top of right hand column,

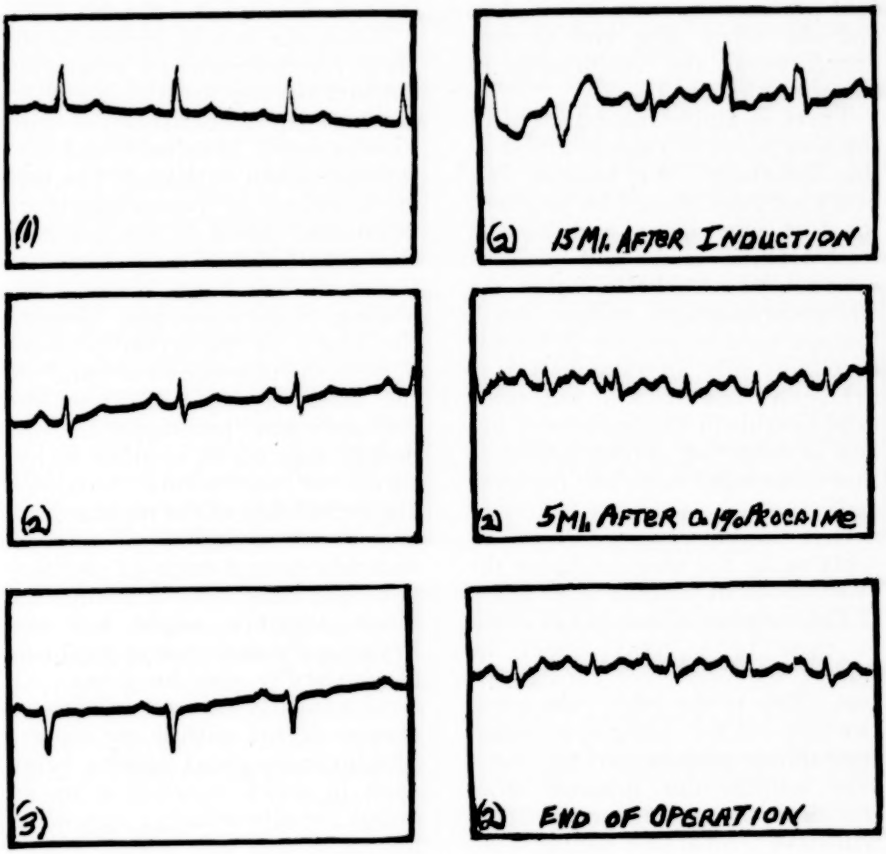
(1)
(2) 15 MI. AFTER INDUCTION
(2)
(2) 5 MI. AFTER 0.1% PROCAINE
(3)
(2) END OF OPERATION

Figure 1

observed 15 minutes after induction, reveals multiple and multifocal premature contractions. The other two leads, one taken five minutes after starting 0.1 per cent procaine by intravenous drip and the other near end of surgery, reveal the restoration of sinus rhythm.

There is ample information for the treatment of cardiac arrest in the literature. We believe that every surgeon should be prepared for this emergency and have a well trained team and plan of action. We also believe that the electrocardiograph and/or oscilloscope used in surgery is important not only in demonstrating arrhythmias which untreated might result in cardiac arrest, but also in detecting cardiac arrest so that massage can be initiated early.

It is the anesthetist's responsibility to let the surgeon know the exact moment trouble is at hand. If the surgeon is not in the chest or close to a great vessel, he cannot see or detect cardiac arrest. This is the job of the anesthetist. The surgeon must reestablish circulation by massage within four minutes from the time of arrest or the brain will have irreparable damage. No time can be wasted. The anesthetist must insure adequate oxygenation and make certain the airway is open. Intratracheal intubation assists in maintaining this. Have a time keeper record events as they occur. The anesthetist should check the electrocardiogram and the radial pulse. If a radial pulse is not felt with each cardiac massage contraction, it is ineffective and this information should be imparted to the surgeon. All this is important to prevent hypoxia. Of equal importance is the prevention of hypercapnia through the use of fresh soda lime to absorb CO_2.

When the use of curare is adverse for the patient's breathing, aid him by bag breathing and do not hesitate to counteract it with Prostigmin®, Tensilon® and Coramine® when indicated. The role of atropine is becoming more debatable. Some do not use it at all. Small doses may stimulate the medullary vagal nuclei stimulating respiration and slowing the heart. A milligram or more of atropine is required to paralyze the blocking vagal effect on the sino-auricular pacemaker.[5] This dosage may result in sinus tachycardia or paroxysmal tachycardia. Irritability of the myocardium may result in the use of myocardial depressants such as procaine and quinidine. As a result the blood pressure might fall and Wyamine® and nor-epinephrine (Levophed®) may be given. All too frequently this set of circumstances occurs with many diverse pharmacological agents being used in quick succession or all going simultaneously; over-medication results and no one knows what is happening for certain; unfavorable results may ensue. Be sure not to over-medicate the patient with barbiturates, digitalis, quinidine, procaine and other drugs. This is as serious as too much anesthesia. Intravenous fluids, plasma or blood, good functioning or well treated myocardium, all with nor-epinephrine will usually overcome hypotension or shock. A short period of shock may be worse than long surgery without shock. A marked change in heart rate may occur with or without a change in blood pressure. When the heart rate becomes alarming it can usually be slowed by 0.15 cc. of a 1:2000 solution of Prostigmin®, intravenously.[6] In some cases there is a rising pulse rate, a fall in blood pressure and a left ventricular strain pattern on the electrocardiogram. One of the rapid acting digitalis glucosides, intravenously, is indicated.[6] At times the blood pressure will fall for no apparent reason with no significant change in pulse rate and no particular change in the electrocardiogram. Nor-epinephrine (Levophed®) is indicated. Four cc. of 1-1000 solution is added to 1000 cc. of isotonic saline, 5 per cent glucose, plasma or whole blood. The average rate of flow is 0.5 to 1 cc. per minute but must be adjusted to the patient's blood pressure response. Ordinarily, it should not be used during cyclopropane anesthesia or with severe myocardial ischemia because of the danger of ventricular fibrillation. A bradycardia that does not respond to atropine is usually an ominous sign.

Knowledge of all the tools available and good judgement on how to use them is indispensable in today's handling of major surgical problems.

SUMMARY

The subject of prevention and treatment of cardiac emergencies during anesthesia has been presented in an outline manner. The importance of learning everything that can be known of the patient's physical and mental status has been stressed. Anesthetic agents, general care and attention for emergencies such as a serious cardiac arrhythmia that may develop during surgery have been discussed.

A well trained team, a prepared plan of action and availability and proper use of the necessary tools assure success in surgical cases that otherwise might result in failure.

REFERENCES

1. Eisaman, J. L.; Caylor, H. D., Jackson, C. E., and Roe, H.: Cardiac arrhythmias during anesthesia. A comparison of cyclopropane and pentothal nitrous oxide-ether anesthesia. Am. J. Surg. **86**: 23-28, July 1953.

2. Griffith, G. C.: Personal communication.

3. Taylor, I. B.: Cyclopropane anesthesia; with a report of the results of 41,690 administrations. Anesthesiology **2**: 641-653, Nov. 1941.

4. Carter, F. S., and Eisaman, J. L.: Cardiotoxic action of intravenous procaine. J. Indiana M. A. **44**: 369-373, May 1951.

5. Goodman, Louis and Gilman, Alfred: The Pharmacological Basis of Therapeutics. (New York: MacMillan Co., 1941).

6. Phelps, M. L., and McAfee, J. C.: Problems of anesthesia for surgical procedures within the chest. J.A.M.A. **152**: 1004-1006, July 11, 1953.

Surgery of the Heart and Great Vessels: Anesthesia

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Since November, 1944, when Dr. Blalock performed the first subclavian to pulmonary artery anastomosis for tetralogy of Fallot, this procedure has been carried out on 1275 patients at The John Hopkins Hospital. Although, in 1944, surgery of the heart and great vessels was done only rarely, today such operations make up a regular part of the surgical practice in hundreds of hospitals. As a result, anesthetists everywhere are becoming increasingly interested in the problems peculiar to this field of surgery.

Surgical attempts to correct cardiovascular disease were begun many years ago. Among the early workers were Cushing and Branch,[1] who reported experimental operation on the heart valves of dogs in 1907. In 1914 Carrel and Tuffier carried out similar work. Allen and Graham, in 1922, developed a cardioscope with which they were able to cut the mitral valve on dogs.

In 1923, Cutler attempted to relieve mitral stenosis. Three years later Souter first inserted a finger in the left auricle and digitally explored a mitral valve. The present day operation for the relief of mitral stenosis is very similar to his technic.

Gross of Boston ligated the first patent ductus arteriosus successfully in 1938. In October, 1944, Crafoord of Sweden resected a coarctation of the aorta. The first coarctation operation performed in the United States was done by Gross in 1945.

There were other workers in this field in the early part of this century but the aforementioned surgeons gave the real impetus to cardiac surgery. In 1948, Brock of London reported a method of direct attack on the pulmonary valve for the treatment of pulmonary valvular stenosis, and also a method of direct attack for infundibular stenosis. That same year reports were made of the successful surgical relief of mitral stenosis by Smithy, Bailey, and Harken.

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[1] Nanson, Eric M.: The Present Scope of Cardiac Surgery, New Zealand Med. Jr., Vol. L: No. 280, 537-551, 1951.

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The development of chemotherapeutic agents and antibiotics, as well as the use of blood transfusions in preventing and treating shock, has played an important part in making cardiac surgery possible. Improvements in anesthetic technics have also had a large role in this development. Thoracotomy and cardiotomy are now considered relatively safe and practical procedures.

Patients with cardiovascular disease who are amenable to surgery, and in whom a fairly good prognosis can be expected from surgical intervention, may be divided into two groups: Congenital and acquired heart disease.[2]

Congenital heart diseases include:

1. Patent Ductus Arteriosus
2. Coarctation of the Aorta
3. Tetralogy of Fallot
4. Pure Pulmonic Stenosis
5. Tricuspid Atresia
6. Anomalies of the Aortic Arch
7. Pulmonary arteriovenous fistula
8. Anomalies of the pulmonary venous return
9. Auricular septal defects

Acquired heart diseases include such conditions as:

1. Wounds of the heart
2. Mitral Stenosis
3. Constrictive pericarditis
4. Traumatic Arteriovenous Fistula
5. Arterial Aneurysm
6. Aortic Stenosis
7. Essential hypertension

Time permits only a brief discussion of the more common types of heart disease treated by surgery at the present time.

PATENT DUCTUS ARTERIOSUS

In this anomaly there is a persistent patency of the ductus between the pulmonary artery and the aorta, which is normally obliterated with the first respiration after birth.

These patients are acyanotic and usually asymptomatic, but they are in danger of developing left-sided heart failure or subacute bacterial endocarditis and, therefore, operation is usually advisable.

The typical case is not difficult to diagnose. It is typified by a "machinery-like" murmur, heard most clearly over the second interspace to the left of the sternum. In view of the technical procedures now available for obtaining a permanent closure of the ductus, with a low mortality, Blalock believes the operation is indicated for all children beyond two or three years of age who have a patent ductus. While the operation in children and young adults is not dangerous, the procedure is more difficult and dangerous in the older age group.

COARCTATION OF THE AORTA

In this malformation there is an abnormal constriction of the major blood vessel. The constriction is usually located just below the left subclavian artery. This constriction reduces the blood pressure in the lower extremities with resultant hypotension in the legs and increases the blood pressure in the upper extremities and neck vessels, with resultant hypertension in the arms. These patients develop collateral circulation around the point of constriction and it is this extensive collateral circulation which makes it possible to occlude the aorta while performing the anastomosis.

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[2] Blalock, Alfred: A Consideration of Some of the Problems in Cardiovascular Surgery. Jr. Thor. Surg. Vol 21: No. 6, 543-571 June 1951.

There is rarely any difficulty in the diagnosis of coarctation of the aorta. The patient usually remains asymptomatic until around twenty years of age. At this time he may notice cramps in the lower extremities on exertion, or symptoms of hypertension, such as blurred vision, headache or occasionally, loss of consciousness. Early diagnosis is based on the presence of a systolic murmur usually heard at the aortic area, associated with hypertension in the upper extremities and hypotension in the lower.

In approximately 80% of these patients, chest X-ray shows notching of the ribs. This is due to the increased blood flow through the intercostal arteries which serve as collateral vessels in by-passing the point of constriction. The treatment of the adult type of coarctation, the common form in which the constricted segment is short, consists of the excision of the stenotic segment and an end-to-end anastomosis of the aorta. In the infantile type, the constricted segment is too long to permit end to end anastomosis. This type necessitates the use of an arterial homograft or the subclavian artery for the anastomosis.

Tetralogy of Fallot

Most patients with pulmonary stenosis or atresia, belong in the most commonly observed cyanotic type of congenital heart disease known as tetralogy of Fallot. The components of the complex are: (1) pulmonary stenosis or atresia, (2) interventricular septal defect, (3) dextroposition or over-riding of the aorta, (4) right ventricular hypertrophy.

The symptoms and signs associated with the disease are direct consequences of the disturbance of normal circulatory dynamics. Cyanosis, the outstanding sign, results from the constant mixing of unoxygenated blood from the right ventricle with systemic blood. This mixing of venous and arterial blood occurs because the pulmonary stenosis produces an increase in right ventricular pressure, causing a shunt of venous blood through the interventricular septal defect or into the over-riding aorta. The stenosis also reduces the pulmonary blood flow. Polycythemia is usual, with red blood counts ranging from normal to 12 million. The hematocrit may be as high as 80 instead of the normal of 45. The oxygen saturation of arterial blood varies from 12 to 90%. The Blalock-Taussig operation for relief of this condition consists of a shunt type procedure designed to create an artificial ductus arteriosus between the systemic and pulmonary circulations, thus bypassing the point of stenosis. This permits some of the incompletely oxygenated blood of the systemic circulation to be recirculated through the lungs. An anastomosis is constructed between the proximal end of the divided subclavian artery or the aorta itself - a Potts type anastomosis - and the side of the right or left pulmonary artery.

This allows systemic blood to pass through the created ductus to both lungs. Occasionally, because of anatomical anomalies, it is not possible to anastomose the end of the subclavian artery to the side of the pulmonary artery. Under such a condition the proximal end of the subclavian artery is anastomosed to the distal end of the pulmonary artery. This permits systemic blood to reach only one lung and only partial benefit may be expected.

PURE PULMONIC STENOSIS

This is usually referred to as valvular stenosis occurring without an interventricular septal defect. However, in approximately 70% of these patients the foramen ovale has been kept patent by the high right ventricular pressure. In these patients the stenosis is not really isolated or "pure". If the foramen ovale is patent the unoxygenated blood may spill over into the left auricle in response to the high pressure that is transmitted back to the right auricle from the right ventricle. In advanced stages this right to left shunt is so pronounced as to produce obvious cyanosis, which leads to confusion with tetralogy of Fallot. A large group of patients who were formerly thought to have a tetralogy of Fallot have since been found to have "pure" pulmonic stenosis.

Taussig has stated that the incidence of pure pulmonary stenosis is about one-tenth that of tetralogy of Fallot. Life expectancy is rarely more than twenty-six years. Since there is no interventricular septal defect and consequently no unoxygenated blood in the aorta (when the foramen ovale is closed), there is no reason to carry out a shunt type of operation. Such an operation will increase the blood supply to the lungs but it will also provide an intolerable strain on the heart. Since the primary, and in many cases, only defect is obstruction of the pulmonary valve, the logical approach to the condition is a direct attack on the valve with division of the obstruction. This not only provides physiologic improvement but also restores the heart to an approximately normal anatomic condition.

The cardiac disorders that have been referred to are all congenital. There is, however, an even larger group of patients incapacitated by acquired heart disease. Rheumatic fever is the chief offender. Stenosis of the mitral and aortic valves is the most serious sequela of rheumatic fever. It is probable that the use of antibiotics and other available agents will reduce the incidence of infection and thereby decrease the incidence of mitral stenosis. Unfortunately, many attacks of rheumatic fever in childhood are never diagnosed. For this reason it is probable that there will continue to be a moderate number of cases of mitral stenosis despite preventive and therapeutic advances.

In general, the patients with acquired heart disease, by the time they reach the attention of the surgeon, are older than those with congenital heart disease. The right heart has been subjected to strain for many years. Most of these patients have had one or more episodes of pulmonary edema, right-sided heart failure and, in some instances, hemoptysis.

Not all patients with mitral stenosis are candidates for surgery. Andrus believes the indication for commissurotomy—division of the stenosis valve—is as follows: Symptoms and signs of pulmonary engorgement; dyspnea and cough on exertion; paroxysmal dyspnea; pulmonary edema, or frank hemoptysis. Blalock states the most promising cases are those without enlargement of the left ventricle or prominent enlargement of the left auricle, which indicates mitral insufficiency and contributes significantly to the patient's disability. A small degree of insufficiency does not contraindicate valvulotomy. Prognosis is always unfavorable when there is evidence of increasing stenosis.

This discussion will serve as a brief illustration of the type of patient the anesthetist encounters in a cardiovascular clinic. Now let us consider the anesthesia.

Adequate sedation and premedication are of the utmost importance. The inability of the cyanotic patient to meet an increased demand for oxygen and the additional strain on the heart caused by fear and apprehension in the non-cyanotic or older patient, necessitate careful consideration in the selection of premedication. Of equal importance is the time of administration in relation to the administration of the anesthetic. Cooperation between the nurses, surgeons and anesthetists is essential.

Our preference, in most instances, continues to be morphine and atropine in appropriate doses. In some instances, especially where hypotension is feared, parenteral nembutal may be substituted for the morphine. The congenital cyanotic patient has a high tolerance for morphine. The reason for this is not completely understood. It may be the lowered metabolism permits the available oxygen to be more efficiently utilized. We compute the morphine dosage on the basis of 1 mgm. for each 5 kilograms of body weight for children. Adults receive 10 to 12 mgm. depending on their age, weight, condition, and degree of apprehension. The atropine dosage is 1/20 that of the morphine, up to 0.6 mgm. There is a tendency by some to omit atropine as a preliminary drug for cases of mitral stenosis. This is done in the hope of avoiding tachycardia, which may predispose to pulmonary edema. We have omitted atropine in a small number of cases in which tachycardia was present preoperatively, but we do not omit it routinely. The premedication should be administered one hour prior to the induction of anesthesia.

ANESTHESIA

We feel the anesthesia should be kept simple and uncomplicated. Excellent results are being obtained throughout the country with a variety of agents. The agent or combination of agents with which any one group is most familiar and which gives them their best results, should be the agent or agents and technic of choice. For infants and children our practice is to employ cyclopropane/oxygen, with or without ether for the induction, until intubation has been accomplished. If the operation is to be a direct attack on the heart valve, the anesthesia is maintained with an ether/oxygen mixture following intubation, in an effort to reduce the occurrence of arrhythmias. If the surgical procedure is to be on the vessels adjacent to the heart, as for instance, the Blalock-Taussig or Pott's operation, cyclopropane/oxygen, with or without small amounts of ether, is the maintenance of choice. There is a definite amount of psychic trauma associated with cardiac surgery for the adolescent and adult patient. Our aim is to make the induction period as rapid and smooth for this group as is compatible with safety. For these patients we use pentothal sodium with a sufficient amount of muscle relaxant to facilitate intubation. Maintenance is then continued with cyclopropane/oxygen, ether/oxygen, or even pentothal/nitrous oxide/oxygen. In the past, d-tubo-curare has been the relaxant agent of choice. We now prefer succinylcholine because of its rapid and ultra-short action. The muscle relaxant is administered only during the induction period. We do not feel that curare is innocuous to heart action. Some years ago we frequently administered small amounts during surgery, when it was difficult to obtain quiet respirations. Invariably, prompt bradycardia developed following the injection. We felt this occurred too often to be purely coincidental and discontinued its use except during the induction period.

We feel inhalation anesthesia is to be avoided for induction of the patient with mitral stenosis. Not infrequently some degree of pulmonary edema is present. This may contribute to a prolonged period which in turn increases apprehension and results in increased pulmonary edema. A vicious cycle is effected.

Our first patient in the mitral series was unwisely anesthetized with cyclopropane. After a stormy period of fifteen minutes, sufficient relaxation was obtained to permit intubation. Immediately a massive quantity of frothy fluid was ejected through the catheter and the patient had regained consciousness. Pentothal was quickly administered and therapeutic measures instituted to control the edema. One hour and a half later the operation was performed without any further difficulty and the patient made an uncomplicated recovery. This incident probably would not have occurred if pentothal had been the induction agent. Since then, pentothal has been employed almost routinely for the induction of anesthesia for mitral valvulotomy.

No pressor drugs are administered to offset anticipated hypotension. The routine use of intravenous procaine hydrochloride or procaine amide was practiced in the early cases of our series in the hope of decreasing arrhythmias. These substances did not appear to be of any material benefit as a routine practice and their use was discontinued. In our experience, arrhythmias, unless present preoperatively, are transient and usually well-controlled by adequate pulmonary ventilation. We believe the emphasis should be placed on efficient pulmonary ventilation with a high percentage of oxygen in the mixture, rather than on the employment of a variety of drugs. Maintenance is always at a very light level of anesthesia. Patients undergoing cardiac surgery require surprisingly small amounts of anesthetic agents. The To and Fro absorption technic has been the method of choice. We prefer to assist the respiration constantly by manual compression of the breathing bag. Occasionally controlled respiration is employed. The non-rebreathing technic, using the Digby Leigh or Stephen-Slater valve, has been recommended, especially for infants and small children. This technic offers the advantages of: Reduced dead space; prevention of carbon dioxide accumulation; and heat retention is kept at a minimum. Our anesthesiologists, at one time, preferred to employ the non-rebreathing technic. The nurse anesthetists find it easier to maintain smooth, satisfactory anesthesia with adequate assistance of the respirations with the To and Fro technic.

Should bradycardia develop, the lungs are inflated with 100% oxygen. This alone will usually restore cardiac tone and rate promptly. If the bradycardia persists, 1 or 2 cc. of 10% calcium chloride is injected into the heart muscle with good results. Should cardiac standstill develop a routine cardiac resuscitation program is immediately instituted.

In our experience the two points which deserve the most emphasis from the anesthetists point of view are: (1) the maintenance of the lightest possible plane of anesthesia at all times and (2) direct and continuous visual observation of the heart action by the anesthetist throughout the operation, from the time the chest is opened until it is closed. Direct observation of the heart has more significance to the experienced observer than the measuring of blood pressure or counting of pulse.

Heat retention is a constant source of concern, especially for the small patient. We continue to apply an ice cap to the canister, summer or winter. The small patient is also placed on a water-cooled rubber mattress. Using these measures hyperthermia has been avoided in the great majority of patients.

REPLACEMENT THERAPY

Replacement therapy is carefully adjusted to the requirements of the individual patient. The patient with congenital cyanotic heart disease usually has an excessively high red cell count and hematocrit. Plasma was, until recently, administered to this group unless excessive blood loss occurred during surgery. At present dextran has replaced plasma. Whole blood is, of course, given to the non-cyanotic or anemic patient during surgery, and in the cyanotic group when massive hemorrhage occurs. Caution is observed not to overload the vascular bed as this would put a further load on an already overburdened heart.

A cut-down is made on the saphenous vein at the ankle for all cardiac procedures and two cut-downs are done for operations for resection of a coarctation of the aorta. This is to provide a

(Continued on page 293)

Safety in Anesthesia

John B. Dillon, M.D.*
Los Angeles

With the advent of better understanding of the physiology of disease and trauma, and with the development of antibiotics and other therapeutic methods, the type of surgery done and the types of patients on which it is done have been widened greatly. Surgical mortality is slowly being reduced to those patients with inoperable conditions and those who die incident to anesthesia.

Both the professional and lay press have become more concerned with anesthesia. Twenty years ago, the accepted answer to a death in surgery was, "The patient's heart could not stand the strain," and this was true enough. Today, a further question is being asked—"Why?" This question is sometimes extremely difficult to answer in a satisfactory way. This interest and concern by both the profession and the lay press is reflected in the increase of malpractice actions relating to anesthesia. In this community, for instance, malpractice insurance rates for anesthesia are very close to the highest of all types of malpractice insurance written.

The important position of anesthesia in the patient's general welfare is becoming increasingly apparent, and this is entirely proper. Anesthetic technics are, and must be, modified in the light of an appreciation of the advances in physiology and pharmacology in order to increase the safety of anesthesia for the patient.

Anesthetic agents presently available, and adjuvant drugs, comprise the most potent and rapid-action group of drugs used in medicine today. It is inevitable that, in the employment of these agents, there will be a certain hazard, morbidity and mortality. It is absolutely necessary, however, since it is now possible to begin to assay effects and toxicity of these drugs, that those who administer them be thoroughly cognizant of the responsibility they undertake and train themselves accordingly, so that morbidity and mortality can be reduced to the absolute minimum. For it is truthfully said that the patient's life is in the anesthetist's hands. The following, in a general way, are

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some of the problems that enter into the application of safer anesthesia:

PREMEDICATION

It is a too common experience that patients premedicated by surgeons are overmedicated. This is due, in general, to one or two causes, or both.

a. Lack of appreciation of the necessity for individualization in the dosage of drugs used for premedication, as evidenced by the common "standard orders" which are, more often than not, written for patients in their 20's or 30's, and yet given to patients either much younger or much older; or they are written by the surgeon who has a desire to render his patients senseless before they are exposed to anesthesia. This may, under certain circumstances, be desirable, but more often it is not.

b. Preanesthetic respiratory and circulatory depression add to the strain of anesthesia. It is certainly recommended that the very severely depressed patient should not be anesthetized, and in such circumstances, anesthesia and surgery should be delayed until such a time as the patient is not severely depressed.

INDUCTION

Exposure of a patient to hypoxia during induction must be avoided. Utilization of low oxygen-containing mixtures is not justified, nor is it necessary. It is easy and practical to induce patients without hypoxia with today's diversified technics. It is true that certain few patients become serious airway problems during induction, due to physical peculiarities. Such airway problems and incident hypoxia must be controlled, and it is mandatory that anyone who assumes the responsibility of administering anesthesia be able to institute any remedial measures necessary.

Oxygen should always be given with agents used for the induction of open drop ether, and oxygen should be given with open or semi-open ether technics. There is no excuse for not using it. Addition of oxygen does not prolong induction. It may require more agent to make up for the depressing effects of the hypoxia that accompany the failure to use oxygen. Oxygen should always be given before intratracheal intubation, if other than cyclopropane-oxygen or ether-oxygen are used for induction. The appearance of cyanosis is not part of a properly induced anesthesia.

MAINTENANCE

Surgical anesthesia should be maintained no deeper than is demanded for the surgical procedure. The patient should not be kept in a state of anesthesia that produces a profound muscular relaxation for longer than is absolutely necessary for the surgical requirements. It is essential that respiration be supported by complementation or supplementation during periods of profound relaxation, no matter how produced, since the muscles of respiration are affected in the production of relaxation. The fact that the muscles of respiration are more resistant to relaxation or deep anesthesia does not mean that they are not affected.

Under no circumstances should pentothal, or other short-acting barbiturates, be used alone for surgical anesthesia. The use of these drugs in dental procedures is unique in this respect, for the pain of dental procedures appears to be both qualitatively and quantitatively different from other surgical pain.

It is mandatory that avenues for replacement therapy be provided during surgery. In any major surgery, no less than a No. 18 gauge needle should be used for intravenous solutions, so that blood may be given readily, should it be necessary. It is no time to think of starting an intravenous when it is absolutely needed to bring a patient out of circulatory depression. The route of administration should be available and used to prevent the occurrence of such depression. In chest surgery and in neurosurgery, two intravenous, 18 gauge needles should be in place before surgery starts.

It is inadmissible that an anesthetist be put in a position where respiration and circulation cannot be adequately controlled at all times during the course of surgery.

Termination of Anesthesia

An anesthetic should be so administered that a patient is in full respiratory control and stable from a vascular standpoint at the end of surgery, and no anesthetic should be considered concluded until a patient has full reflex control. The airway must be open and free. The tracheal bronchial toilet should always be done when possible, and obviously should be done following an intratracheal anesthetic. No anesthetist should permit himself to be rushed to another case at the expense of a patient who is not entirely stable and with adequate ventilatory capacity.

Problem of Cardiac Arrest

Recently, a great deal of emphasis has been placed on the treatment and occurrence of cardiac arrests—too little on its prevention. It is believed by many that cardiac arrest, or fibrillation, is an indication of pre-existing hypoxia, with few exceptions, preexisting fear being one. A study on this subject at an Eastern hospital showed that approximately 50% of all cardiac arrests occurred either during induction or at the termination of anesthesia. The study of cardiac arrests at a hospital in this city showed that fear was a common denominator for cardiac arrests during surgery. It is felt that the liberation of adrenalin within the body sets the stage for fibrillation or arrest, and that it is triggered by the anesthetic or by supervening hypoxia. The practical problem that presents itself in this respect is that the over premedicated patient is hypoxic, and that the under premedicated patient is apprehensive. It would appear, therefore, that a considerable consideration should be given to the preoperative preparation of patients in reference to anesthesia, as well as to blood chemistry and fluid balance. Unfortunately, all too often this phase of patient preparation is overlooked.

SUMMARY

Some of the problems in the management of anesthesia have been discussed. It is imperative for patients' welfare that more consideration be given to the preparation and conduct of anesthesia.

1. Premedication must be individualized.
2. Hypoxia must be avoided on induction of anesthesia.
3. Maintenance must be as light as possible and respiratory depression and circulatory depression avoided.
4. Mechanisms for respiratory supplementation and circulatory control must be instituted and available prior to their need.
5. Anesthesia must be so conducted that a patient is not depressed at the termination of surgery.
6. Consideration must be given to the prevention of the occurrence of cardiac arrest.

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The Anesthetist's Responsibility for Malposition

Forrest E. Leffingwell, M.D.*
Los Angeles

The position in which a patient is placed while anesthetized may well become the deciding factor in his recovery; it may produce complications and traumatic lesions which exceed, in seriousness and discomfort, the disease for which he is undergoing surgery; at best, it may cause alterations in physiological processes which, although not immediately detectable, are not in the best interest of the patient and may result in a stormy recovery. In general, the gross positioning of the patient is dictated by the desire to produce maximum exposure and optimum operating conditions as directed by the surgeon. Hoary custom and common practice too frequently are the deciding factors in the absence of the surgeon's stated preference. Some of the most malevolent positions to which we adhere fall into this category and would not survive if we gave close scrutiny to either their damaging effects or the lack of logic behind their application. For example, how can one logically expect that forcing a gallbladder lift up against a fairly rigid spine and bony rib cage will raise the liver and gallbladder to easier accessibility? None can deny that such a malposition puts a very severe strain on the ligaments of that portion of the spinal column, while falling blood pressure and other evidences of physiological insult frequently testify to respiratory embarrassment, and compression or stretching of the vena cava.

An anesthetized patient has been deprived of the privilege of speaking for himself to disclose pains, strains and discomforts; yet the sequellae of such may extend into the immediate postoperative period or longer to plague him with unnecessary suffering. Malpositions which trespass on physiologic processes may be recorded by the patient's reflex responses, but these will be evident only to a trained observer.

It becomes obvious immediately that the anesthetist who has

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placed the patient in this helpless situation has the obligation of protecting him from this same helplessness. To do this he must be aware of the conditions which beget trauma and be able to recognize evidences of physiologic abuse.

PHYSIOLOGIC ABUSES

It has been correctly stated that the primary function of an anesthetist is to insure that his patient maintains adequate pulmonary exchange. The deleterious effects of oxygen want and carbon dioxide excess are too well known to require discussion here. Not so well recognized is the fact that certain positions may so restrict respiratory activity that adequate ventilation becomes impossible. It should be pointed out that in this discussion we are dealing with individuals who are under the influence of drugs and anesthetic agents which inherently depress respiration and circulation, many times to a degree that has all but erased normal reserves even in the best of positions. In such a patient, any further interference will produce inadequacies of ventilation or circulation which can only result in deterioration.

Case and Stiles[1] have shown that vital capacity is markedly affected by certain positions. They found the least alteration in the sitting position. Placing the subject in the dorsal recumbent, prone and lateral positions decreased vital capacity by as much as nine to twelve per cent. Raising the gallbladder or kidney lift decreased function by another two or three per cent. They found the lowest readings in Trendelenburg and lithotomy positions. These studies were made on healthy subjects between the second and seventh decades of life. *THEY WERE UNSEDATED AND UNANESTHETIZED.* It is significant to note that the older subjects showed more profound changes than did the younger ones.

Numerous other investigators [2;3;4]; report that commonly used operative positions bring about various undesirable changes in healthy unanesthetized patients. Miller[5;6] feels that malpositions may be the cause of many surgical fatalities.

With but minor variations, the studies of all investigators point to very definite conclusions:

The weight of the abdominal viscera against the diaphragm in Trendelenburg position definitely restricts the excursion of that organ, in direct proportion to the degree of incline. Increased age, which carries with it some normal limitation in the motions of the rib cage and general loss of vigor in muscular effectiveness, further impairs respiratory movements. Add the depressant or paralyzing effects of anesthetic agents and sedatives to all of this and we have produced a situation which could well sacrifice the life of the patient unless the anesthetist is fully cognizant of the additive insults and assists him through.

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[1] Case, Evelyn H., and Stiles, J. A.: The effect of various surgical positions on vital capacity. Anesthesiology, **7**:29-31 (Jan) 1946.

[2] Sokalchuk, A., Ellis, D., Hickcox, C. and Greisheimer, E. M.: Pulmonary function as affected by operative position. Anesthesiology, **10**:577-584 (Sept) 1949.

[3] Slocum, H. C., Hoeflick, E. A., and Allen, C. R.: Circulatory and respiratory distress from extreme positions on the operating table. Surg., Gynec. & Obst., **84**:1051-1058 (June) 1947.

[4] Stephen, C. R.: The influence of posture on mechanics of respiration and vital capacity. Anesthesiology **9**:134-140 (Mar) 1948.

[5] Miller, A. H.: Surgical prone posture. J.A.M.A. **108**:185-187, 1937.

[6] Miller, A. H.: Posture in anesthesia, New England J. Med., **218**:385-386, 1938.

Lithotomy position in which the thighs are acutely flexed so as to bring external compression to bear upon the abdomen acts to the same end.

For better exposure during operations upon the kidney the patient is often placed in a position of acute lateral flexion. The movements of the rib cage on the upper side are restricted through tension of the overlying tissues. The ribs on the under side are rendered immobile by compression over the ridge of the table. Raising the kidney lift adds to this immobility, compresses the abdomen, and brings about other harmful effects which will be discussed below. Adequate exposure can be obtained by much kindlier treatment. Placement of the patient so that the apex of the break in the table brings its support over the crest of the ilium produces almost the same degree of lateral flexion without the severe compression effect on the under side. When this position is used the kidney lift will rarely be necessary.

Expansion of the chest cavity during a normal inspiration is achieved by movement of the ribs in an upward and outward direction which results in anterior displacement of the sternum. Anything which limits or immobilizes this anterior movement of the sternum will restrict the motions of the ribs to the same degree and thus interfere with the inspiratory act. This is the means by which respiration is hampered by placing the patient in the prone position with his body resting flat upon chest and abdomen. With every breath he must lift the weight of his torso in order to increase the anteroposterior diameter of his chest cavity. The only other means of increasing the capacity of the thorax is by downward movement of the diaphragm. This is also impeded in the flat prone position by compression of the abdomen. The prone position can be improved, and more freedom of movement of the sternum and abdomen secured by supporting the patient with rolls under the iliac crests and shoulder girdle. The normal movements of the sternum and ribs may also be hampered in any position by application of adhesive or other strapping across the chest. Patients should never be anchored in this way. All strapping for the purposes of immobilizing the body in a given position should exert its force on the crest of the ilium and the shoulder point.

The jack knife position is a modified prone, which is subject to all the evil influences of the flat prone, plus the added one of abdominal compression over the apex of the jack knife. The principles of support mentioned above apply equally to it with the additional precaution of bringing the apex of the table to apply its support to the iliac crests.

Malposition impairs circulatory function in two principle ways:

1. By constricting and compressing large vessels.
2. By altering the hemodynamics of circulation.

On many occasions the author has observed severe blood pressure drops, which have occurred under pressure of a kidney or gallbladder lift, immediately return to normal upon lowering these lifts. While the influence of hypoxia cannot be entirely discounted, the prompt response which resulted when compression was relieved would indicate an obstruction to venous return to the heart by stretching or external pressure upon the vena cava. For those surgeons who still feel that pressure applied posteriorly will improve accessibility of the gallbladder, a small pad, such as a folded towel placed under the lower part of the rib cage on the right side only, will be just as effective and much more rational.

The cape of cyanosis, which spreads over the head, neck and shoulders of a patient in sharp Trendelenburg position, is commonly observed. This represents a stagnation of circulation in all areas drained by the superior vena cava and will not clear up under the administration of higher oxygen concentrations alone. Prolongation of this position cannot help but exert whatever baleful influences hypoxia has on the vital centers of the medulla. Furthermore, the slow venous return from the upper part of the body retards cardiac filling.

Hodgkinson and Rood[7] have studied the effects upon hemodynamics from tilting anesthetized patients in various positions. They showed that moderate Trendelenburg position appears to aid return circulation from the pelvis and abdominal areas, as evidenced by a lowering of venous pressure in the ovarian vein. However, placing the patient in an exaggerated Trendelenburg position (more than fifteen degrees) resulted in an increased venous pressure. They suggest that this effect is probably due to reflexes set off by overloading the heart by excessive venous return.

Any change in the incline of the patient's body or large portion thereof, such as the lower extremities, will result in hemodynamic alterations. The anesthetist must have knowledge of the principles involved if he is to accept his full responsibility for protecting his patient. He must know that in steep degrees of reverse Trendelenburg (and to a greater extent in upright positions) venous return to the heart is markedly impeded and large quantities of blood will pool in the dependent areas in a patient under anesthesia. He will help to prevent this by wrapping the lower extremities as a part of his routine pre-anesthetic preparation. The same pooling of blood occurs in the vessels of lower extremities, which are suddenly lowered after having been elevated in lithotomy position for long periods.

TRAUMATIC HAZARDS

In addition to protecting his patient against physiologic trespass, the anesthetist must guard him from the traumatic hazards of malposition and mishandling. These vary all the way from minor muscle or ligament strains to the production of pathologic lesions involving soft tissues or nerves. Although many times these incidents are in no way related to the anesthetic or its administration, the anesthetist cannot entirely escape the responsibility for allowing them to occur, if such could have been prevented by his knowledge of, and attention to, prophylactic measures. The patient is at least partially positioned and prepared while the surgeon is scrubbing and gowning, and the anesthetist is in a much more favorable position to observe and detect potentially dangerous practices.

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[7] Hodgkinson, C. P. and Rood, R. C.: The influence of body posture upon arterial and venous blood pressure in gynecologic surgery. J. Am. A. Nurse Anesthetists, **21**:157-162.

Many of the minor strains of ligaments, muscles and joints can be avoided by asking the question, "Is this position natural and would it be comfortable to a conscious patient?" Of course much surgery requires placement in other than "natural" positions, but the deviation in all cases should be as little as possible.

Exaggerated rotation of the head and neck should be avoided. If the operative site is on the lateral or postero-lateral aspect of the head, raising that side of the body with sand bags to a forty-five degree tilt will minimize neck torsion. In thus raising one side of the body, it is essential to place sand bag supports under both the shoulder and pelvis; raising one without the other will produce torsion of the spine which may result in just as uncomfortable postoperative sequellae as would be caused by torsion of the neck. Supporting the shoulders with a roll calls for support of the head to avoid hyperextension and dangling. Similarly, the head should not be permitted to hang unsupported when the patient is in the lateral position.

Patients who are maintained for long periods of time in the flat, supine position should have a small pillow placed to support the lumbar curve. Slocum[8] has recently advocated what he terms the "reflex abdominal position." It shows a striking resemblance to that produced by a contour chair and appears to achieve the objective of naturalness in the ultimate degree.

Since the most natural position of the joints of the extremities is either neutral or in slight flexion, care should be taken to see that they are so maintained in the unconscious patient. In all instances, hyperextension must be avoided scrupulously. This may call for a soft pillow under the knees. The larger joints of the extremities may suffer injury if the extremity is allowed to drop over the edge of the table or suddenly fall from a support such as the stirrup. When turning patients over, or rolling from table to stretcher, free the legs and arms from all restraints and place the arms in such position as to preclude twisting or crushing. Of course the head must be supported in all such maneuvers.

Forcing the thighs into extreme abduction to straddle a pair of widely set stirrups may strain the adductors or hip joints in an elderly patient. Such a patient should be accommodated by placing the legs inside the stirrup posts which, in most instances, will provide adequate exposure. Flexing the thighs acutely, besides producing the respiratory embarrassment previously alluded to, may cause severe sacro-iliac strains and trauma to the sciatic nerve from excessive stretching.

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[8] Slocum, H. C.: Unpublished data contained in paper read before the Alumni Postgraduate Convention of the College of Medical Evangelists, Feb. 1954.

Skeletal deformities and alterations due to disease should be respected. Never force a partially ankylosed joint or one which shows limitation of function. Elbows which will not straighten should be placed on an armboard in the position they assume, and the forearm should be supported at that angle. Any degree of abduction which will cause pain in the shoulder of a conscious patient should be avoided in that same patient if anesthetized. Patients with dorsal kyphosis or torticollis should be supported with pillows in whatever position is the most natural to them. Not in the same category, but equally important, is the problem of orthopnea. Orthopneic patients should be placed in the position of optimum comfort when awake and kept in that position throughout the surgery. This means that they will probably be half-sitting. The difficult breathing of the orthopneic patient is a physiologic response to pathological changes which will not be banished by the anesthetic.

If the patient's hand is tucked under his body, or otherwise placed out of sight, make sure the fingers are straight and the wrist not cocked. Feet and toes may be similarly injured by the weight of Mayo stands and instrument trays which have been lifted from the floor by raising the table. Decubitus ulcers over the shin bone have been produced in this way.

Meticulous care should be taken to insure that no part of the body is unprotected against pressure from metallic or other hard objects. Heels or Achilles tendon should not be allowed to rest on the metallic surface or hang over the edge of the table. Squeezing an obese patient between stirrup posts without involving pressure on the lateral aspect of the hip may challenge one's ingenuity. The metallic part of a cautery pad or indifferent electrode should rest against soft tissue and not a bony prominence. Patients whose arms are secured at the side by cuffs fastened to the table are in danger of being hung up by the wrists if placed in steep Trendelenburg. Damage may result to the wrist or to a nerve from stretching.

Pressure injuries of the face, though fortunately not too common, can leave disfiguring scars. Face masks strapped on too tightly have produced localized ischemia and subsequent sloughing of skin over malar prominences and the bridge of the nose. Some of the conductive rubber masks have shown a marked tendency to produce pressure burns of this nature. Healing may leave a keloid scar or unsightly discoloration. The same situation can result from the corner of a sand bag or other firm object pressing into skin which has little between it and the bone underneath. Patients placed in a face down position in a cerebellar head rest must be checked and re-checked every time the head is moved. No part of the face must be allowed to rest on a hard or rough surface, even as rough as a towel. Pressure burns are very difficult to prevent in this position. The eyes should be closed and covered with a pad well-buttered with petrolatum.

While burns do not often occur in the operating room and are seldom related to malposition, they do happen under circumstances which can well come under the scrutiny of the anesthetist and many of them can be prevented by diligent observation. Contact with the cautery pad should be over large enough surface so that electro-coagulation does not result. Likewise, any bare skin which is in loose contact with any metal object may draw sparks when cautery is in use and produce a burn. If there is any occasion to apply a hot water bottle to an anesthetized patient, the temperature should be well below that which will cause discomfort to a conscious person.

The most likely sources of burns are those produced by chemicals. Though not of such frequent occurrence as in the days when more irritating and caustic antiseptic solutions were used, they still produce burns in an occasional patient whose skin is hypersensitive. The common practice of allowing antiseptic solutions to run down the groin or between the arm and body has resulted in a few severe burns to contiguous surfaces. Similar burns have resulted from painting over adhesive tape. Although such instances are rare and can probably be laid to individual idiosyncracy, the patient involved derives little comfort from the fact.

Eyes should be protected from irritants such as soap and other prepping solutions. Permitting the eyelids to remain half open may result in drying of the cornea and ulcers which will cause the patient more pain and discomfort than any part of the operation. The so-called "ether burns" of the eye following open drop anesthetics are more than likely due to this cause.

The most crippling and damaging lesions which can result from malposition are the peripheral nerve injuries. Although many of them are transient they frequently require weeks or months for recovery and some leave permanent residuals. These injuries are due to prolonged pressure over, or stretching of a nerve, or both. A few moments of routine prophylactic checkup may save some patient weeks of disability.

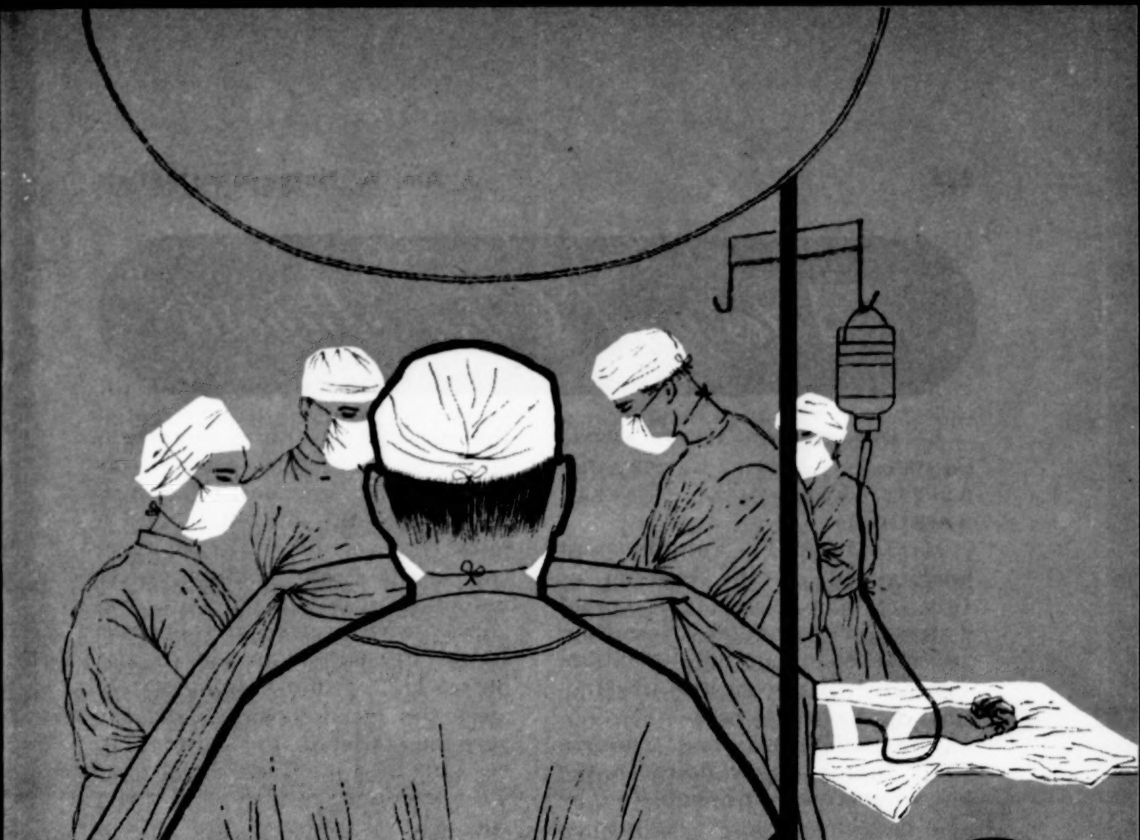
Peripheral nerves of the upper extremity may be damaged by allowing the arm to hang over the sharp edge of the table; from wrist strappings which are too tight; from being suspended by the wrists in Trendelenburg positions; and from the metal stays of a snap-on blood pressure cuff pressing into the ulnar notch. Similarly, nerves of the lower extremity may sustain damage from stirrup posts, straps across the legs (particularly in the lateral position), and pressure from legs being strapped down onto hard rolls in the popliteal space.

Injuries to the brachial plexus most frequently occur from stretching, though pressure from a poorly padded shoulder brace can be damaging. This is particularly true if the arm is abducted while the weight of the patient is pressing hard against a shoulder brace on the same side. Severe stretching damage has resulted from nothing more than maintaining an arm outstretched on a board at too great an angle for a prolonged period of time.

CONCLUSIONS AND SUMMARY

Much of this presentation dwells on what seem to be obvious facts and elementary exhortation, but much of the tragedy we see in life stems from simple, preventable situations in which intelligent well-informed individuals find themselves, through their own or someone else's carelessness. All of the accidents mentioned above have occurred, many of them in the hands of experienced, though temporarily unthinking, anesthetists. A few simple practices will minimize them:

1. Keep positions natural and avoid all exaggerations.
2. Deviate from the natural only through necessity, not custom.
3. Assist all deficiencies of respiration and correct those of circulation.
4. Remember, the price of continued safety is eternal vigilance.



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1. Helrich, M.; Papper, E. M., & Rovenstine, E. A.: *Anesthesiology* 11:33, 1950. 2. Stephen, C. R., & Martin, R.: *North Carolina M. J.* 12:501, 1951. 3. Phillips, H. S.: *Anesth. & Analg.* 32:56, 1953.

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Notes and Case Reports

A Report of Hypercapneic Convulsions with Respiratory Arrest Following General Anesthesia.

Mrs. Mary T., age 49, was hospitalized on the evening of July 16, 1953, in acute abdominal distress with nausea, vomiting, and marked abdominal distention. Tentative diagnosis was obstruction of the small bowel.

The patient had had a fourth stage thoracoplasty done on the left several years previously. She also had had a radical right mastectomy for carcinoma and that operation was followed later by deep X-ray therapy for a post-adenocarcinoma of the pelvis. In addition, the patient had recently had shock therapy for a depressive mental illness. Chest examination revealed that there were no breath sounds on the left, and the breath sounds were very much diminished on the right. Her temperature was 99° F. Hemoglobin was 10.5 grams, red cell count, 3,310,000, and the white cell count, 10,600. Urinalysis showed a trace of albumin.

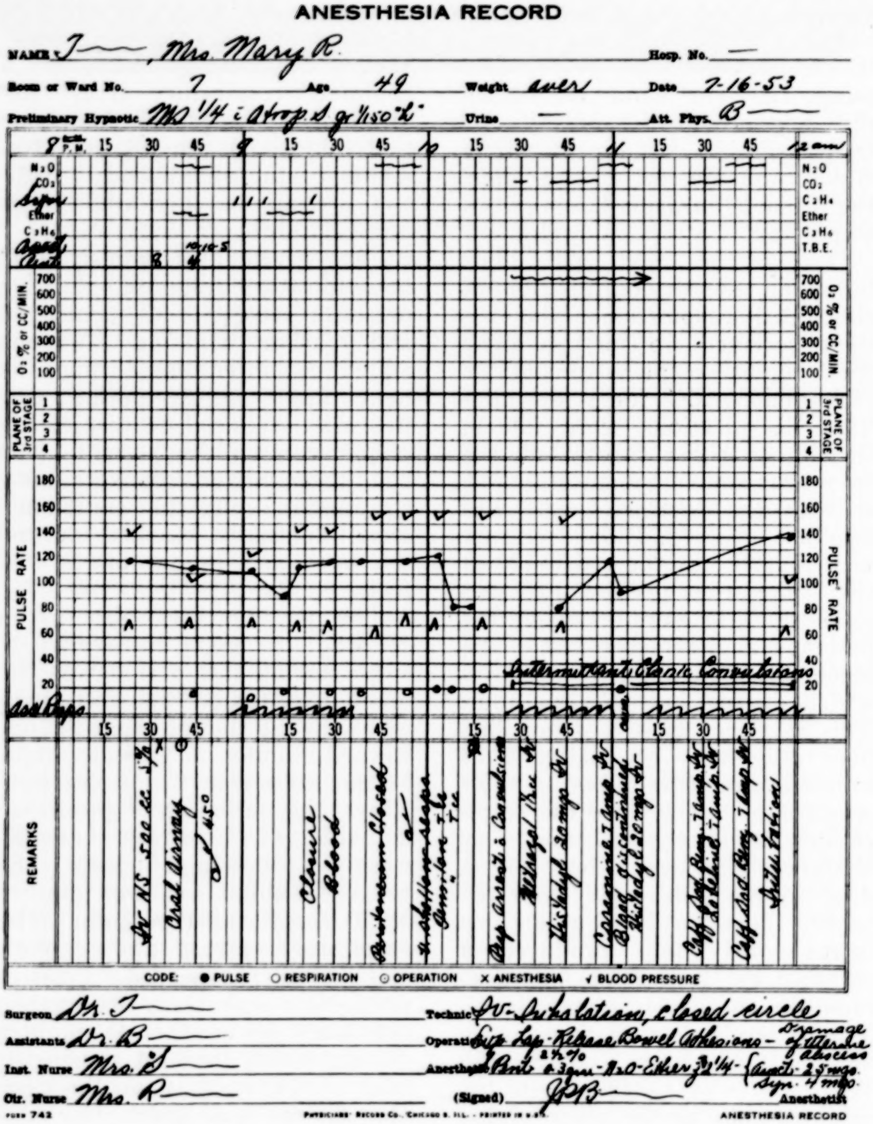
The patient was given Morphine Sulfate gr. 1/4 with Atropine Sulfate gr. 1/150 by hypodermic as a pre-anesthetic medication one hour before being taken to surgery at 8:30 p.m. Induction was with Pentothal Sodium 2 1/2%, 0.3 gm. followed with Nitrous Oxide - Ether inhalation with the closed technic during maintenance. Although the patient was quite apprehensive, the induction period was smooth and uneventful with a Blood Pressure of 140/72 and a pulse rate of 120. The incision was made eight minutes after induction was started; an oral airway was inserted at this time. Blood Pressure was 112/76 and pulse 112. Almost immediately after the incision was made, the surgeon ordered the patient to be put into a steep 45 degree Trendelenburg position. During the next 15 minutes the patient was given a total of 25 mgs. of Anectine in three divided doses in the intravenous tubing. (An intravenous infusion of 500 cc. 5% Dextrose in Normal Saline had been started in the left arm before the induction.) Relaxation was still not as complete as the surgeon desired, although the respirations were already depressed and assisted respirations were required. Blood Pressure was 124/72 and pulse 112. During the next half hour the patient was entirely on controlled respirations in order to maintain a good color. Syncurine 4 mgs. was given intravenously during this time to maintain adequate relaxation for the surgeon. Several small bowel adhesions were released and a large uterine abscess was drained. This abscess was extremely difficult to drain and expose because the previous deep X-ray therapy had caused much scarring of the pelvic tissue.

Fifteen minutes before the peritoneum was closed, controlled respirations were discontinued and the patient was breathing on her own at 20 times per minute. Blood Pressure was 144/72 and pulse 120—the same as before the beginning of surgery. A transfusion of 500 cc. of whole Blood was started at this time, the previous infusion having been completed.

During the next half hour following the discontinuation of controlled respirations, the patient received no further anesthetic agent except Nitrous Oxide - Oxygen 50-50% for a few minutes. Respirations were full in the lower right chest, but the color was somewhat questionable so that the Nitrous Oxide was also discontinued. Blood Pressure was 152/70 and the pulse 120 at the time the peritoneum was closed. Eight minutes later, the patient was completely out of the steep Trendelenburg position in which she had been for one hour. This leveling off of position had been done very gradually. Five minutes after the operating table was completely leveled the respirations suddenly became very shallow. This was followed by a drop in the pulse rate from 120 to 84. Tensilon 2 cc. was given intravenously and produced a satisfactory return or improvement in the respiratory volume. The patient was then thoroughly aerated with 100% oxygen and the mask was taken off in ten minutes time at the close of the operation. Respirations were 20 per minute, pulse was still 82, Blood Pressure still 152/74, and the color was satisfactory. The patient seemed to be in the lower plane of second stage anesthesia and in satisfactory condition.

Ten minutes later, after being moved to the litter for transfer to her room, the patient suddenly became cyanotic and went into a complete respiratory arrest followed by generalized clonic twitchings and convulsive movements of the entire body. Positive pressure oxygen was begun immediately and Metrazol 1 1/2 cc. was given intravenously with no respiratory response. Positive pressure oxygen was continued and Carbon Dioxide 5% was added at intervals during the next thirty minute period; but this also produced no respiratory response from the patient. The convulsive movements were fairly continuous, and the twitchings were especially marked around the eyes and in her hands and feet.

The patient was also given Histadyl 20 mgs. intravenously twice during this period, and the blood transfusion was discontinued with the thought that the untoward reaction or episode might be due to a transfusion reaction. Later, the patient was given one ampoule of Coramine intravenously following which she breathed alone for ten minutes at 20 respirations per/min. The respiratory arrest and convulsions then reappeared. Positive pressure respirations were then continued for another 45 minutes during which time, at the insistence of the internist on the case, the patient was given two ampoules of Caffeine Sodium Benzoate and one ampoule of Lobeline intravenously. (The internist was attributing the reaction to the curare agents that had been



ANESTHESIA RECORD

NAME T——, Mrs. Mary R. Hosp. No. —

Room or Ward No. 7 Age 49 Weight aver Date 7-16-53

Preliminary Hypnotic MS 1/4 c̄ Atrop. S gr 1/150 (H) Urine — Att. Phys. B——

8 a.m. p.m.
15
30
45
9
15
30
45
10
15
30
45
11
15
30
45
12 am
N2O
CO2
C3H4
Ether
C3H6
T.B.E.
10-10-5
O2 % or CC/MIN.
700
600
500
400
300
200
100
PLANE OF 3rd STAGE
1
2
3
4
PULSE RATE
180
160
140
120
100
80
60
40
20
Intermittent Clonic Convulsions

REMARKS

Iv NS 500 cc 5%

Oral Airway

∫ 45°

Closure

Blood

Peritoneum Closed

a

[illegible]

Tensilon ī cc

" ī cc

Resp. arrest c̄ Convulsions

Metrazol 1 cc IV

Histadyl 20 mgm IV

Coramine ī amp IV

Blood discontinued

Histadyl 20 mgm IV

Caff. Sod. Benz. ī amp. IV

Lobeline ī amp. IV

Caff. Sod. Benz. ī amp IV

Intubation

CODE: ● PULSE ○ RESPIRATION ⊙ OPERATION X ANESTHESIA √ BLOOD PRESSURE

Surgeon Dr. T——

Technic IV-Inhalation, closed circle

Assistants Dr. B——

Operation Exp. Lap.-Release Bowel Adhesions - Drainage of Uterine abscess

Inst. Nurse Mrs. S——

Anesthetic Pent. 2½% 0.3 gm - N2O - Ether ℥ 1/4 - {Anect. 25 mgs. Syn. 4 mgs.

Ctr. Nurse Mrs. R——

(Signed) JPB—— Anesthetist

FORM 742

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ANESTHESIA RECORD

Anesthesia record prior to hypercapneic convulsions.

given during the anesthesia; the anesthetist felt that there was some other cause.) The patient was also intubated during this period. The convulsions stopped again soon after the last Caffeine Sodium Benzoate injections, but the respirations continued in arrest for almost another hour. Positive pressure oxygen with intermittent addition of Nitrous Oxide 1:1 was continued. The pulse rate had risen from 84, at the onset of the convulsive episode, to 140 after the last Caffeine injection. Blood Pressure was now 104/70.

Finally, almost three hours following the onset of the episode, the patient was again breathing on her own and "bucking" on the intratracheal tube. Extubation was performed and the patient was fully awake soon afterwards. She was then returned to her room in satisfactory condition. Her post-anesthetic recovery from then on was strictly uneventful, and her post-operative recovery was good. Her condition was excellent the following day. She was discharged from the hospital on the 7th post-operative day. Her family later told the surgeon that the patient had also done poorly under anesthesia when she had had the radical mastectomy done.

Later, after the case had been carefully reviewed, the untoward reaction of respiratory arrest and muscular twitching were attributed to an excess accumulation of Carbon Dioxide in the body tissues over a long period of time, thus causing a very severe hypercapnia. This severe hypercapnia was felt to have been due to the factors of the fourth stage thoracoplasty on the left, a healing radical mastectomy on the right, and acute abdominal distention, plus the added insult of a steep 45 degree Trendelenburg position for one hour. These factors resulted in an inefficient aeration of the lungs. The retained accumulation of Carbon Dioxide caused an unrecognized severe acid-base imbalance. This acid-base imbalance also accounted for the difficulty encountered during anesthesia in maintaining a good color. The anesthetist was also relying too heavily on a new canister of soda lime and was led into a false sense of security of having complete CO_2 control.—**J. Pauline Benefiel, R.N., Indianapolis.**

Abstracts

Gillan, J. G.: Two cases of unilateral blindness following anaesthesia with vascular hypotension. Canad. M.A.J. **69**:294-296 (Sept.) 1953.

"The day has passed when the junior member of the hospital staff administered the anaesthetics. Anaesthesia has risen to its proper place as a specialty. With greater complexity of equipment has come the opportunity to help the surgeon more by improving the condition under which he operates. One of these aids is the use of drugs to reduce haemorrhage.... All the exponents of the technique, in spite of enthusiasm, issue warnings that there are certain possible dangers .... Visual symptoms on administration of hexamethonium orally in hypertensive patients, have been noted.... Two cases are presented where irreversible eye changes occurred associated with vascular hypotension during major surgical procedures.

"Case 1. L.Z., aged 26 years, was under observation at the Oshawa General Hospital for a renal complaint.... On October 29, 1952, an operation was performed on the left kidney.... The anaesthetic was by Gillies method, using a pentothal induction with Novocain and Nupercaine mixture for the spinal block. The blood pressure dropped from 120 to 70 mm. of mercury in 20 minutes, and was maintained at this level for one hour and five minutes. The pressure rose to 100 mm. mercury after one hour and forty minutes. No pressor drug was required. Good oxygenation was maintained throughout. On recovery of consciousness it was discovered that there was loss of vision in the left eye.... The diagnosis of central retinal artery occlusion was made. There has been no restoration of vision since, and pallor of the optic nerve is now becoming apparent.

"Case 2. E. R., aged 48, white female, was examined on October 31, 1953, at the Oshawa General Hospital. She had undergone an operation on the previous day for bowel obstruction. She was found to have a metastatic carcinoma from a primary at the hepatic flexure, and a large resection of the bowel was carried out. The right ovary, which was also involved, was removed. The anaesthetic was by Gillies method using a Novocain and Nupercaine mixture, as in the previous case. The systolic blood pressure prior to the anaesthetic was 165, which dropped in fifteen minutes to 55 mm. mercury. The pressure remained at this level for one hour and forty minutes. After two hours it dropped to 50 mm. mercury, when Methedrine 10 mgm. was given. The pressure rose to 100 after two hours twenty minutes from start of operation. On return to the ward she reported that she could not see with the left eye .... No actual arterial occlusion was found. The patient died on the fourth day after apparently making good progress. Permission for a postmortem was obtained, but did not include the head, although this was requested. It was, therefore, not possible to obtain the pathological background of the clinical findings ....

"A review of the pertinent literature was attempted to discover if blindness had been recorded previously in association with induced hypotension .... A record of permanent eye damage was not found in the literature. A review of the technique employed in the above cases was made by the anaesthetic staff. It appears that new equipment had been ordered, and only partially supplied. Thus new rubber head straps were used in conjunction with an old face mask. It is probable that these exerted undue pressure on the left eye. Pressure on an eye during anaesthesia is not an uncommon cause of unilateral blindness, although it is not stressed in books upon the subject .... It is emphasized that the retinal artery is an 'end-artery', and that blood enters the eye against the pressure of the intraocular tension. That this equilibrium is easily upset is proved experimentally by the transient blurring of vision which occurs after gentle pressure on one eye. When the systolic pressure drops markedly, the excess of the retinal arterial pressure over the intraocular pressure is reduced, so that even slight external pressure may arrest the circulation and allow the occlusion of retinal arteries. That this is not the whole picture is evidenced by the work of Cameron and Burn, who showed that the intraocular pressure also fell when the blood pressure was reduced by hexamethonium in glaucoma patients."

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Book Reviews

Modern Practice in Anaesthesia. Edited by Frankis T. Evans, M.B., B.S., F.F.A.R.C.S., D.A., Consultant Anaesthetist, St. Bartholomew's Hospital, London. Cloth. Ed. 2. 645 pages, 217 figures. New York: Paul B. Hoeher, Inc., 1954. $12.50.

The second edition of this collection of material by a variety of authors follows the original pattern. The names of many of the outstanding British anesthetists are included among contributors to this book. Newer technics, such as controlled hypotension, are included in this second edition. The wealth of information presented by the authors makes this book of great value to persons interested in the field.

A Synopsis of Anaesthesia. By J. Alfred Lee, M.R.C.S., L.R.C.P., M.M. S.A., D.A., F.F.A.R.C.S., Consultant Anaesthetist to the Southend-on Sea Hospital, etc. Cloth. 3d ed., 483 pages. 72 illustrations. Baltimore: Williams & Wilkins Co., 1953. $3.50.

This new edition of a useful book has been completely revised. The style of the earlier editions has been retained but much new material has been added. Those who are familiar with previous editions will welcome this revised version: those who have not yet become acquainted with it have deprived themselves of a stimulating and helpful reference book.

Some Papers on Nitrous Oxide-Oxygen Anesthesia. By Elmer Isaac McKesson, M.D. Edited by K. C. McCarthy, M.D., Toledo, Ohio. Cloth. 180 pages. Privately printed. 1953.

This privately printed edition serves as a memorial to Elmer Isaac McKesson. In reprinting many of his original articles the author has not only made available information of historical value, but has given present day anesthetists an opportunity to learn from the experience of one of the early teachers in the specialty.

Art and Principles of Anesthesia. By Phyllis A. Roberts, R.N., Anesthetist, Greene County Hospital, Jefferson, Iowa, and L.C. Nelson, M.D., Surgeon, Greene County Hospital, Jefferson, Iowa. Cloth. St. Paul: Northland Press, 1954. $3.00.

This book, written by a member of the American Association of Nurse Anesthetists in collaboration with a surgeon with whom she has worked, presents the problems of anesthesia as they have occurred in an extensive practice. One of the outstanding features of the book is the presentation of special problems as they relate to certain types of operations. Nurses who are working in situations similar to those of the author will find this presentation invaluable in solving some of the many problems that are encountered.

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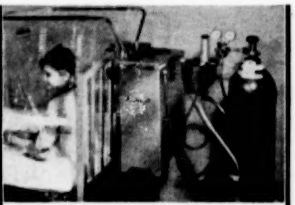

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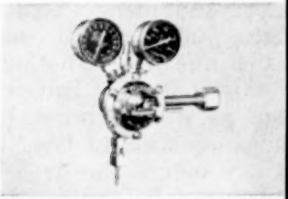

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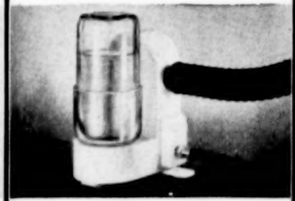

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Legislation

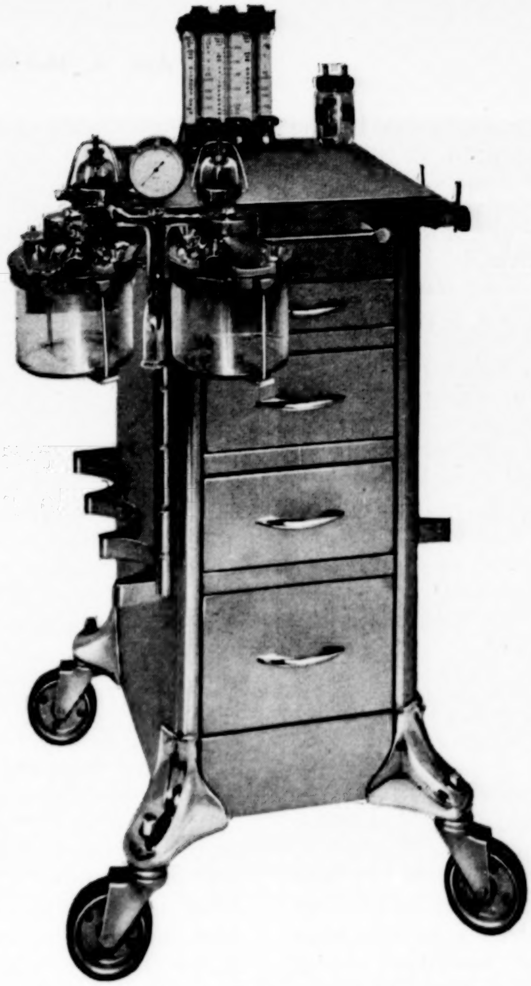
Emanuel Hayt, LLB., Counsel, A.A.N.A.

PHYSICIANS ABSOLVED OF NEGLIGENCE IN ANESTHESIA DEATH OF CHILD—The defendants, one a duly licensed physician and surgeon and the other a duly licensed physician and anesthetist were indicted for manslaughter in the second degree, in that while acting in concert, each aiding and abetting the other, they performed and assisted in the performance of an operation on an infant of ten years of age, and that the death of said infant was by their culpable negligence.

Defendants separately move for an order permitting an inspection of the grand jury minutes, or, in the alternative, for a dismissal of the indictment, upon the ground that the legal evidence received by the grand jury was insufficient as a matter of law to sustain the indictment. The court has examined the grand jury minutes and finds the evidence insufficient to establish culpable negligence within the meaning of the manslaughter statute (People v. Greenwald, 271 App. Div., 800). Furthermore, the record fails to establish compliance with the requirements of section 1041 of the Penal Law as to the criminal agency causing the death and defendants' connection therewith. It is claimed that the child died under anesthesia, that he died from asphyxia, but the cause of death is left to speculation.

In People v. Angelo (246 N.Y., 451), Judge Andrews said at page 457: " 'Culpable' negligence is therefore something more than the slight negligence necessary to support a civil action for damages. It means, disregard of the consequences which may ensue from the act, and indifference to the rights of others. No clearer definition, applicable to the hundreds of varying circumstances which may arise, can be given. Under a given state of facts, whether negligence is culpable is a question of judgment. Ordinarily for the judgment of the jury it is the question whether negligence exists at all. But in the one case as in the other it may become a question of law."

The evidence before the grand jury indicates a difference of medical opinion and judgment as to the choice of the anesthesia used and of the method of its administration, but the court is unable to find any acts or omissions which establish that either of the defendants acted in disregard of the consequences which might ensue from their acts and that they acted with indifference to the rights of the deceased. As was said in People v. Carlson (176 Misc., 230, 232): "Mere lack of foresight, stupidity, irresponsibility, thoughtlessness, ordinary carelessness, however serious the consequences may happen to be, do not constitute culpable negli-



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gence. There must exist in the mind of the accused at the time of the act or omission, a consciousness of the probable consequences of the act, and a wanton disregard of them."

While the court fully appreciates the tragedy involved, it is constrained to hold as a matter of law that the evidence does not establish beyond reasonable doubt that the defendants were guilty of that degree of negligence which may be deemed "culpable negligence", rendering the defendants criminally liable.

Motion granted to the extent that the indictment herein be dismissed. Orders dismissing the indictment signed.

(People v. Herman Wolff and Theodore Rogers, County Ct., Queens County, Farrell, J., N.Y.L.J., June 18, 1954, p 12, col. 2.)

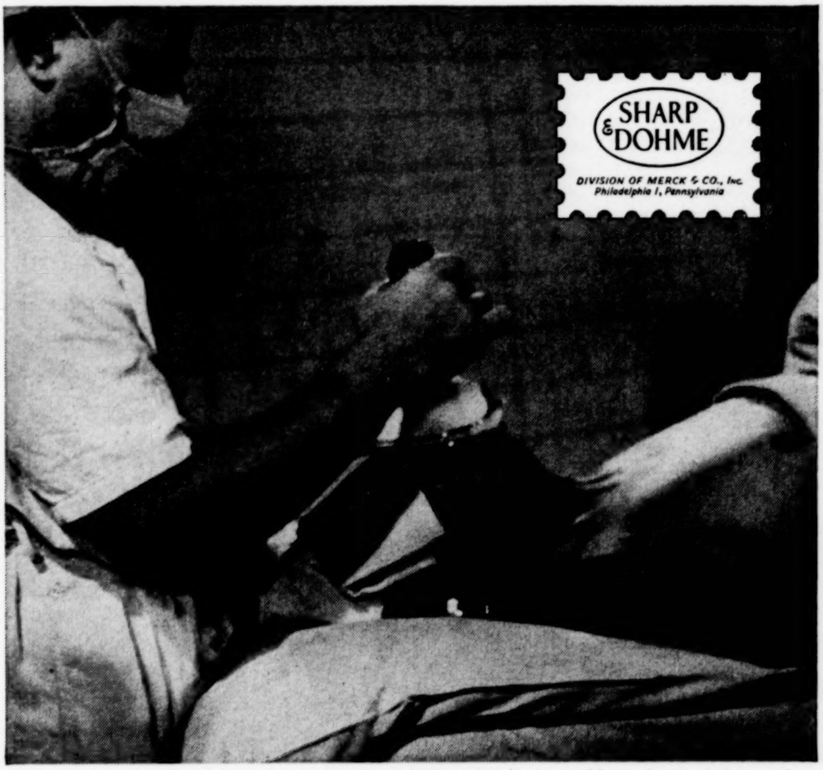
Injured Infant's Claim Not Barred By Statute Of Limitations—The child was injured in the course of its birth. It was not until he was taken to another doctor more than a month after birth that the condition being sued for was determined to be the result of the physician's alleged negligence.

The statute of limitations in the state of California permits actions by infants who are viable at the time of injury, but such actions must be brought within six years from the date of birth.

The statute of limitations does not ordinarily begin to run while the physician-patient relationship continues and only commences to run from the date of discovery of the wrongful act, or the date when by the exercise of reasonable care the patient should have discovered the wrong act. In the present case the patient was not aware of the wrongful act until more than a month after his birth, and during this period the statute of limitations did not begin to run. It is therefore possible for the child to bring this action six years and one month after the date of his birth.

(Myers v. Stevenson, 3 Commerce Clearing House, Neg. Cases 2d series 937-Calif.)

Court Affirms Dismissal Of Death Action Of Newborn Burned In Bassinet—In this action, the administrator of a newborn infant, sues to recover for the intestate's conscious pain and suffering and for its wrongful death, which resulted from the burning of the bassinet in which the infant lay in the nursery in defendant's hospital. The evidence was that the infant's temperature had been subnormal and that, following the customary procedure, a lamp had been placed over its wrapped feet to warm it. After feeding the infant, the student nurse then in charge, noticing that the infant felt cold to her touch, sought to bring its temperature to normal by replacing the lamp over its wrapped feet. However, instead of adjusting the lamp to the usual distance of six inches from the blanket she adjusted it three inches therefrom, which caused the fatal fire. Plaintiff appeals from the order setting aside the verdict in his favor, directing a verdict in favor of defendant and dismissing the complaint, and from the judgment entered thereon. Judgment affirmed with costs. No opinion. Appeal from order dismissed, without costs. No such order is printed in the record. Wenzel,



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COUNCIL ACCEPTED

Acting P.J., MacCrate and Schmidt, JJ., concur. Beldock, J., concurs on the dismissal of the appeal from the order but dissents and votes to reverse the judgment and reinstate the verdict, with the following memorandum: The infant was born normal and healthy. The testimony is that there was no necessity for any medical care for this infant. She was in the custody of the hospital for safekeeping until the mother was well enough to go home. The hospital breached its duty of safekeeping when the student nurse negligently placed the electric bulb too close to the blanket covering the child. Any negligent act which caused injury to the child while in the sole custody of the hospital creates liabililty (see Lederman v. Boulevard Sanitarium, Inc., 263 App. Div., 727, leave to appeal denied, 287 N.Y. 852). In addition the jury might properly find, in view of the fact that no medical treatment of the infant was either requested by the parents or directed by the physician in charge, or was required, that placing the bulb within three inches of the blanket was not in the course of medical treatment of the infant but constituted administrative negligence. Murphy, J., dissents and votes to reverse the judgment for defendant and to reinstate the verdict, with the following memorandum: The proof warrants a finding that it is the defendant's practice to raise temperatures to normal and to place a lighted naked electric bulb within six inches of a newly born infant while it lies wrapped in blankets in a cotton lined bassinet. Such a practice as a matter of hospital routine is manifestly negligent and is of an administrative nature. (Bickford v. Peck Memorial Hospital, 266 App. Div., 875; Iacono v. N.Y. Polyclinic Medical School and Hospital, 296 N.Y., 502). Even if it were of a medical nature, the jury was enabled to find administrative negligence under the general charge of the court, in that the defendant assigned administration of this dangerous practice to a student nurse as the only nurse in charge, and who was permitted to leave the nursery unattended for a substantial length of time. The student nurse placed the naked bulb even closer than the six inches, and the fire occurred during her twenty minute absence (cf. Santos v. Unity Hospital, 301 N.Y., 153)

T. Cadicamo, as adm'r. etc. of Mario Elena Cadicamo, dec'd., op, v. L. I. College Hospital, res., 131 N.Y.S. 2d 287).

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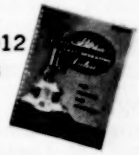

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NURSE ANESTHETIST: For approved 160-bed pediatric hospital. 40-hour week; liberal vacation policy and other benefits. Salary open. Anesthesiologist in charge. Apply: Administrator, Milwaukee Children's Hospital, 721 North 17 Street, Milwaukee 3, Wisconsin.

WANTED: Registered Nurse Anesthetists. Contact, M. Sanders, Director McLaren General Hospital, 401 Ballenger Highway, Flint 2, Mich.

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ANESTHETISTS: A.A.N.A. members. 250 bed general hospital; salary open; automatic increases; laundry provided; forty hour week; no obstetrics; liberal vacation and personnel policy; Social Security. Sutter Hospital, Sacramento, Calif.

WANTED: Nurse Anesthetist for 100 bed Cancer Hospital, active major surgical service, congenial working conditions, excellent equipment, very little call, 40-hour week, salary $448 to $572. Apply Medical Director, Ellis Fischel State Cancer Hospital, Columbia, Mo.

TWO NURSE ANESTHETISTS, above average salary. Medical Anesthesiologist in charge. Inquire C. K. Shiro, Administrator, Montana Deaconess Hospital, Great Falls, Mont. Call at hospital expense.

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WANTED: Nurse Anesthetist to act also as Supervisor of floor on 1 shift at small, 25 bed hospital, average 20 operations per month, another anesthetist to share anesthesia given. Salary $382.50. Location 34 miles south of Memphis, Tenn. All applications treated in strict confidence. Correspondence will be answered promptly. Send COMPLETE history in first letter to the Administrator, Tunica County Hospital, Tunica, Mississippi.

NURSE ANESTHETIST: General hospital, 700 beds; starting salary $4300 per annum, maximum $4800 per annum, $100 yearly increments; vacation and sick time; full maintenance provided; contact A. G. Chmelnik, M.D., Medical Director, The Harrison S. Martland Medical Center, 116 Fairmount Avenue, Newark, N. J.

NURSE ANESTHETIST: For 237 bed general hospital. Additional nurse anesthetist needed. Present staff of five nurse anesthetists, full-time anesthesiologist. Liberal salary and personnel policies. New, modern hospital located in Connecticut, 28 miles from New York City. Write furnishing complete details of training and experience to: Personnel Director, Greenwich Hospital, Greenwich, Connecticut.

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NURSE ANESTHETISTS (3): For 1000 bed University teaching Hospital. 1 month paid vacation, 14 days sick leave, social security, 1 nite call per week. Salary depending on experience. Apply: Dean Eberhardt, Director, School of Anesthesia, Barnes Hospital, St. Louis 10, Missouri.

NURSE ANESTHETIST: Immediate opening for qualified person in well-organized dept. performing 9,300 anesthetics per year. Modern, well-equipped, 400 bed general hospital. Salary dependent upon experience and ability. Liberal personnel benefits. Forward replies outlining work experience, references, and training to: Director of Anesthesiology, The Delaware Hospital, 14th & Washington Streets, Wilmington 1, Delaware.

WANTED: One Nurse Anesthetist for 250 bed approved General Hospital. Rotating shifts with three other Anesthetists. Salary open, partial maintenance provided. Apply to the Director of Nursing Service, E. A. Conway Memorial Hospital, Monroe, La.

WANTED: Nurse Anesthetist for approved general hospital. Full-time Medical Anesthesiologist and 4 nurse anesthetists on staff. Pleasant working conditions. Starting salary $350 per month, annual merit increases. Write: Director of Nursing, Mercy Hospital, Baltimore 2, Maryland.

NURSE ANESTHETIST NEEDED in 152 bed approved hospital. Closed staff. Alternate call with 2 other anesthetists. $400 per month with maintenance and other fringe benefits. Contact: Administrator, Nan Travis Memorial Hospital, Jacksonville, Texas.

REGISTERED NURSE ANESTHETIST: 135 bed general hospital in charming southern city of 18,000 short drive from Gulf of Mexico. Well-qualified surgical staff. Salary range $380-$416 a month commensurate with experience. 4 weeks vacation with pay, sick leave, 2½ day weekend every 4th week. Apply: Administrator, John D. Archbold Memorial Hospital, Thomasville, Georgia.

NURSE ANESTHETIST: Wanted to complete staff of four serving accredited 200 bed hospital. Department directed by Anesthesiologist. Excellent staff of Surgeons. Pleasant working conditions, hours, paid vacation, sick leave, Holidays, Social Security, group health insurance, Retirement plan. Starting salary $350 per month, annual merit increases. Located in the beautiful coulee country on the Mississippi. Apply to E. J. Berg, Manager, Gundersen Clinic, 1836 South Avenue, La Crosse, Wis.

NURSE ANESTHETIST for Oral Surgeons' office. Pleasant working conditions. Regular working hours - 5 and 5½ days alternating weeks. Good future. Send replies to: Box D-18 The Journal, A.A.N.A., 116 South Michigan Avenue, Chicago 3, Illinois.

WANTED: Three Nurse Anesthetists for Anesthesia Department in new wing of 400 bed general hospital. Pleasant working conditions in modern surgical and obstetrical departments. Liberal vacation, sick leave, pension plan benefits. Salary open. Write: Assistant Administrator, Madison General Hospital, Madison, Wisconsin.

NURSE ANESTHETIST: To increase present staff of 10 nurse anesthetists. A.A.N.A. membership or eligibility required. Starting salary $375 per month with yearly increase, plus full maintenance or cash allowance for maintenance, as desired. Private room with bath and telephone in new women's residence. Social Security and private pension plan. Excellent working conditions; surgical and delivery suite air conditioned. 40 hour week including call time. Apply: Marshall Kerry, M.D., Chief Anesthesia, The Reading Hospital, Reading, Pa.

THIRD ANESTHETIST WANTED for approved 184 bed general hospital in city of 25,000. Regulated hours and excellent working conditions. 4 weeks paid vacation. Paid sick leave. $400 monthly plus full maintenance. Living accommodations in nicely furnished nurses' home. Apply to: Trinity Hospital, Minot, North Dakota.

WANTED: Registered nurse anesthetist. Fully approved small, privately-owned hospital located in the Midwest. Salary open, vacations with pay. Excellent working conditions. Contact Mrs. Geraldine Walden, Superintendent. A.C.H. Hospital, Shawnee, Oklahoma.

REGISTERED NURSE ANESTHETISTS: 40 hr. week, permanent positions open for surgery and obstetric departments. Liberal vacation and sick leave policies. Social security, overtime pay, extra pay for night duty. No call duty. Automatic pay increases. Apply: Chief Nurse Anesthetist, Harper Hospital, Detroit 1, Michigan.

NURSE ANESTHETIST for 70 bed hospital planning expansion. Salary open. Vacation, sick leave, and partial maintenance. Apply: King's Daughters Hospital, Brookhaven, Mississippi.

SURGICAL AND/OR OBSTETRICAL POSITIONS available for qualified nurse anesthetists in newly reorganized department. Apply: J. D. Stringham, M.D., Dept. of Anesthesiology, Latter-day Saints Hospital, Salt Lake City, Utah.

WANTED One Nurse anesthetist for 240 bed hospital. Salary open. Partial maintenance provided. Apply: Administrator, Charleston General Hospital, Charleston, West Virginia.

NURSE ANESTHETIST WANTED: 225 bed hospital located in resort city, southern Connecticut. Liberal salary and many benefits. Apply: Personnel Office, Lawrence and Memorial Associated Hospitals, New London, Connecticut.

NURSE ANESTHETIST: 120 bed general hospital. Present staff of two. Terms of employment open. Living-working conditions excellent. 45 miles N.Y.C. Contact: David H. Welsh, M.D., Dir. of Anes., Newton Memorial Hospital, Newton, New Jersey.

NURSE ANESTHETIST: 86 bed general hospital; A.C.S. approval; located in a town of 18,000, two hours drive from Memphis, Tenn. Lovely nurses' home. $400.00 plus full maintenance; alternate working shift with another anesthetist. Apply to: Administrator, Helena Hospital, Helena, Arkansas.

ANESTHETIST - NURSE: 250 bed general hospital; excellent working conditions and personnel policies. Write: Mr. Bert Stajich, Assistant Administrator, Columbia Hospital, 3321 N. Maryland Avenue, Milwaukee 11, Wisconsin.

WANTED: Two nurse anesthetists in new 300 bed, five million dollar hospital. Starting salary $375, after 60 days, $400, then increasing every 6 months. On call once weekly, long weekend every six weeks. Average 44 hour week. Apply: Director of Anesthesia, Methodist Hospital, Texas Medical Center, Houston, Texas.

NURSE ANESTHETIST WANTED: Position open immediately, Butte Community Memorial Hospital, Butte, Montana. Beginning salary, $400.00 per month, plus additional fees for overtime calls. Regular salary increases. Well-equipped department. Three regular Anesthetists. Wire collect, Dr. W. B. Talbot, Administrator.

Muscle Relaxants

(Continued from page 246)

11. Hoppe, J.O.: A new series of synthetic curare-like compounds. Ann. New York Acad. Sc. **54**:395-405, October 1950.

12. Castillo, J.C., and DeBeer, E.J.: The neuromuscular blocking action of succinylchoine (deacetylchoine). J. Pharmacol. and Exper. Therap., **99**:458, August 1950.

13. Foldes, F.F., and McNoll, P.G.: The use of succinylcholine for endotracheal intubation. Anesthesiology **14**:93, January 1953.

14. Foldes, F.F., and Rhodes, D.H., Jr.: The role of plasma cholinesterase in anesthesiology. Anesth. and Analg. **32**:305-318, September-October 1953.

15. Foldes, F.F.; Machaj, T.S.; Hunt, R.D.; McNoll, P.G.; and Carberry, P.C.: Synthetic muscle relaxants in anesthesia. J.A.M.A. **150**:1559-1566, December 20, 1952.

16. Smith, D.L. and Virtue, R.W.: Succinylcholine: a case report and experimental study. Anesthesiology **15**:42-49, January 1954.

17. Beecher, H.K. and Todd, D.P.: A study of the deaths associated with anesthesia and surgery. Annals of surgery **140**:2-34, July 1954.

Surgery of Heart

(Continued from page 262)

ready means of administering large quantities of blood rapidly should hemorrhage occur. With opening of the anastomosis of the aorta, hypotension may be precipitated as the result of the expanded vascular bed. At this time blood is pumped until the blood pressure has become stabilized. Thus far we have not found it necessary, nor do we believe it desirable, to employ arterial transfusions for cardiac surgery.

CONCLUSION

We would like to emphasize the employment of these simple anesthesia technics: maintenance of efficient pulmonary ventilation rather than the employment of a variety of drugs, which tend to confuse the issue; maintenance of a light level of anesthesia; and, last but not least, constant visual observation of the heart action by the anesthetist.

The TWENTY-FIRST QUALIFYING EXAMINATION for membership in the American Association of Nurse Anesthetists will be conducted on Saturday, May 14, 1955.

The deadline for accepting completed applications, including the transcripts, is April 4. If application without transcript is received too close to the deadline, the application may not be considered by the committee in time for the candidate to be scheduled for the May examination. Notice of eligibility will be mailed about April 10.

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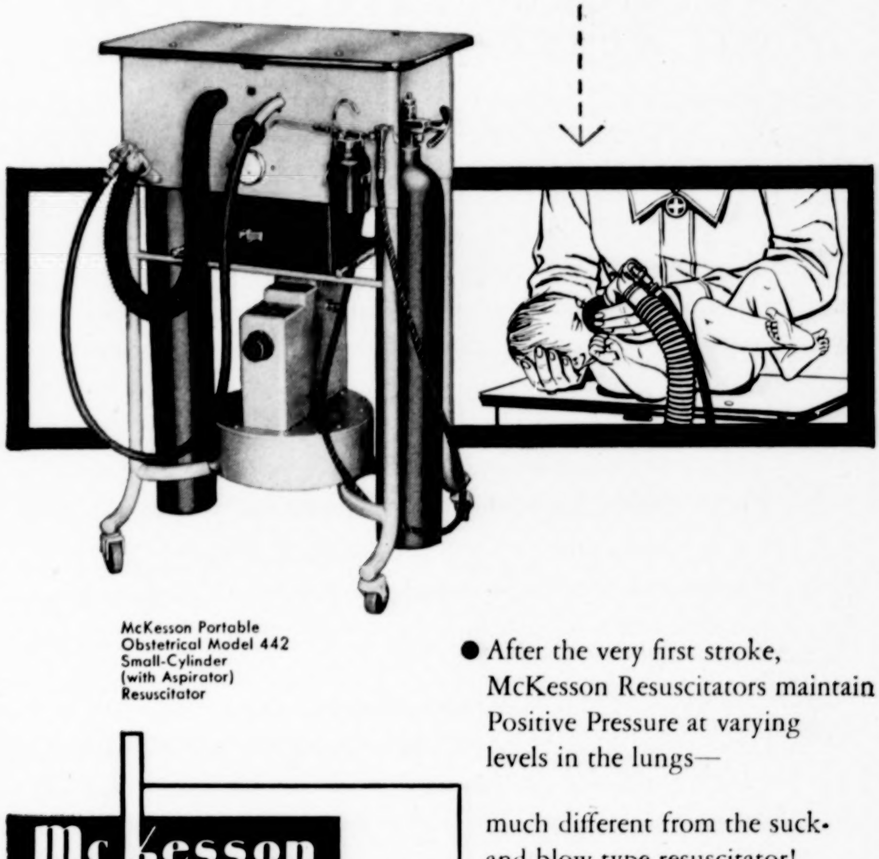

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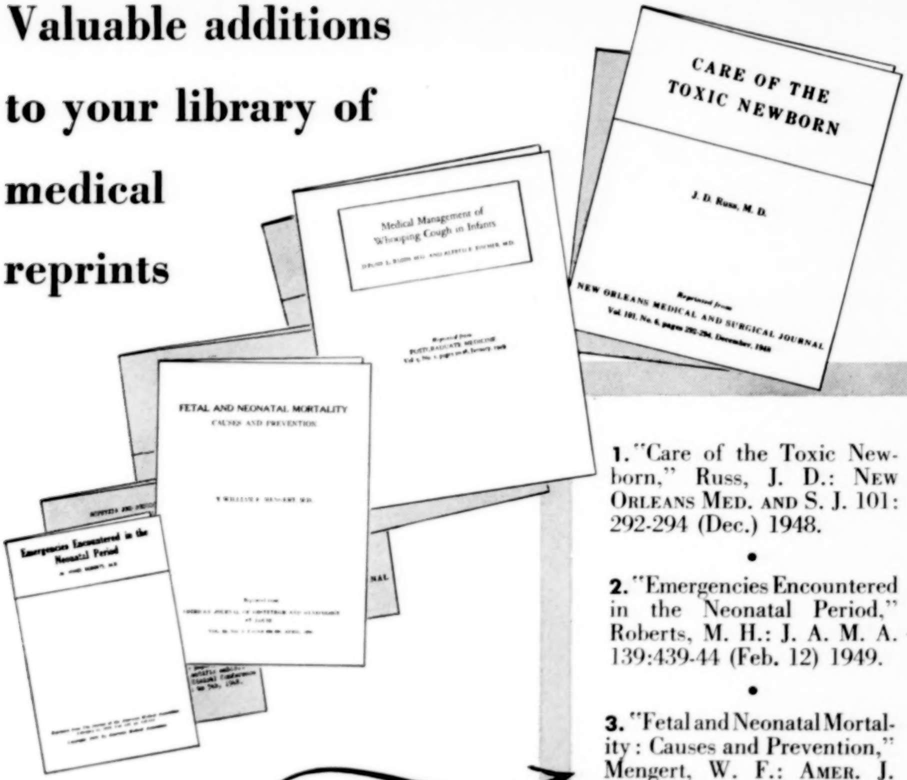
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Vol. 101, No. 6, pages 292-294, December, 1948
Medical Management of Whooping Cough in Infants
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POSTGRADUATE MEDICINE
FETAL AND NEONATAL MORTALITY
CAUSES AND PREVENTION
Emergencies Encountered in the Neonatal Period

1. "Care of the Toxic Newborn," Russ, J. D.: New Orleans Med. and S. J. 101: 292-294 (Dec.) 1948.

●

2. "Emergencies Encountered in the Neonatal Period," Roberts, M. H.: J. A. M. A. 139:439-44 (Feb. 12) 1949.

●

3. "Fetal and Neonatal Mortality: Causes and Prevention," Mengert, W. F.: Amer. J. Obst. and Gyn. 55:660-668 (April) 1948.

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4. "Medical Management of Whooping Cough in Infants," Kohn, J.L. and Fischer, A.E.: Postgrad. Med. 5:20-26 (Jan.) 1949.

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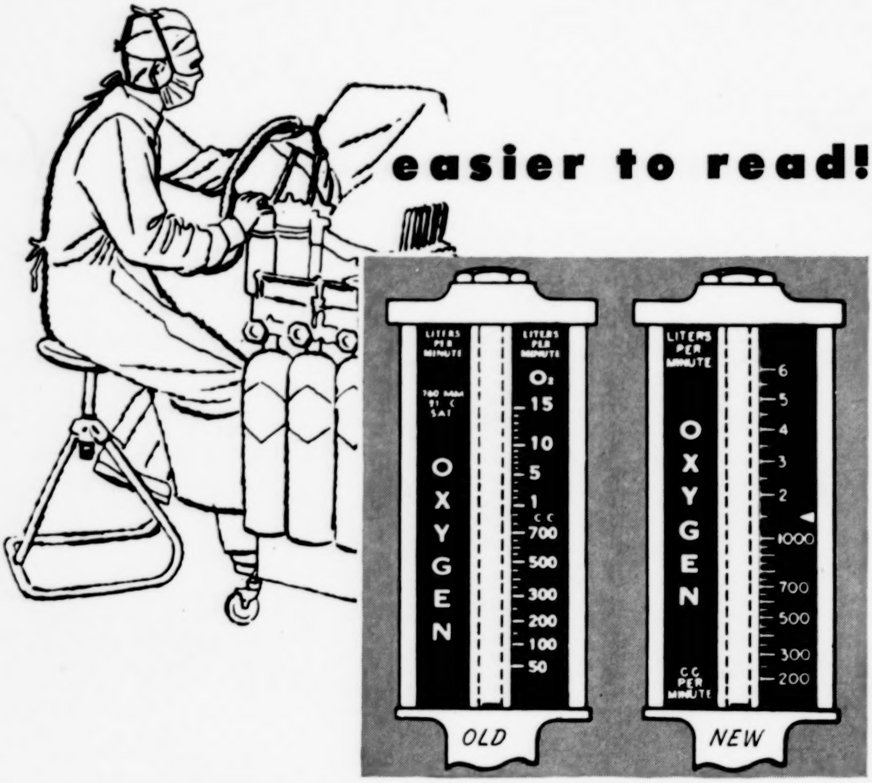
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